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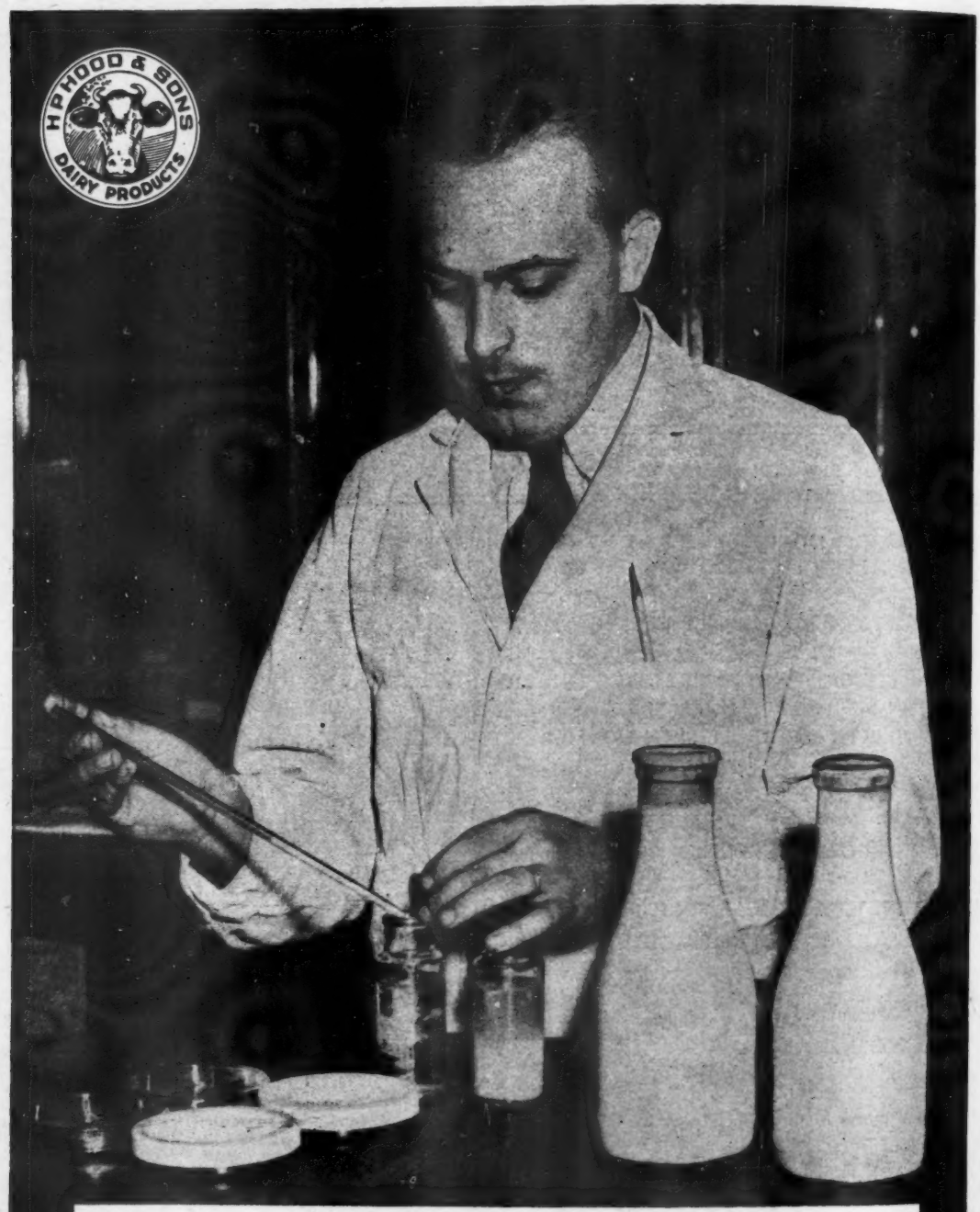

THE best milk available in any community is that which is subjected to the most rigid and exact controls . . . on the dairy farms and at the milk plants.

H. P. HOOD & SONS

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PEPTIC ULCER AMONG SOLDIERS IN THE MEDITERRANEAN THEATER OF OPERATIONS

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TECHNICIAN (5TH GRADE) HENRY WEINBERG, MED. DEPT., A.U.S.

AT THE beginning of the war, it was thought that peptic ulcer would present a serious military medical problem. This has evidently been the case in Army hospitals and induction centers in the United States. Owing to rejections at induction and discharge from the service of patients in Army hospitals in the Zone of the Interior, the disease was not seen in the Mediterranean theater of operations with the frequency expected, particularly after the first year. At the Sixth General Hospital in North Africa, however, to which a considerable proportion of patients were sent for evacuation to the United States, an opportunity was presented between March, 1943, and January, 1944, to study an unusually large number of patients with peptic ulcer.

Several reports[1–3] have appeared regarding the incidence and clinical features of this disease as seen in Army hospitals in the United States and in overseas theaters. In the present report are presented data in 200 consecutive cases among enlisted men. In certain aspects, the disease in these cases appears to differ from that noted among recent inductees in Army hospitals in the United States.

We have been interested in two main aspects of the problem — the purely clinical aspects and the psychosomatic features. In connection with the latter, certain data have been obtained bearing on the question of whether the nervous tension of Army life is a factor in either the etiology or the course of the disease. In addition, observations have been made regarding the personality differences between patients with peptic ulcer and those with chronic psychogenic dyspepsia; these observations appear to be of practical value in differentiating the two disorders.

STATISTICS

The large number of soldiers with chronic epigastric distress admitted to the Sixth General Hospital in a period of ten months may be arbitrarily divided into those with peptic ulcer and those with dyspepsia not caused by ulcer, after excluding the cases in which the distress was caused by cholecystitis or some other abdominal disorder. Two hundred and twenty-four patients with proved ulcer were admitted to this hospital; 24 of them were officers and are not included in this study. The exact number of patients with non-ulcer dyspepsia is not known, because many men had been on various services and had been discharged with varying diagnoses, particularly when the primary disorder was surgical, temporarily submerging the importance of the epigastric distress. A minimum figure, however, may be obtained from the total number of gastrointestinal x-ray examinations that were performed, since before disposition most of the patients with chronic indigestion were examined by x-ray in this hospital even though previously examined elsewhere. With the ulcer cases subtracted from the total number receiving gastrointestinal x-ray examinations, it is estimated that there were between 500 and 600 cases of non-ulcer dyspepsia. Thus, the ratio of ulcer to non-ulcer cases is about 1:3 — a higher one than has been noted in most Army hospitals in the United States. The chief reason is that this hospital, being at the tip of a funnel for evacuation to the Zone of the Interior, received a considerable proportion of all ulcer patients in the theater. This ratio does not, therefore, signify that the incidence of peptic ulcer among soldiers with chronic epigastric distress is higher in the Theater of Operations than in the Zone of the Interior.*

*Since this paper was written, we have had the opportunity to study patients with chronic dyspepsia at the Sixth General Hospital in Italy between July 1, 1944, and November 1, 1944. This hospital received patients from the front a few days after evacuation. Between November 1, 1944, and May 1, 1945, 350 patients with chronic dyspepsia were studied at a gastrointestinal center in the Fifth Army area. One hundred and eighty-three of these patients were studied roentgenologically by Captain S. M. Wyman, M.C., A.U.S. Among these, 113 consecutive patients were x-rayed and gastroscoped to study the problem of "gastritis," as well as to test the accuracy of the history alone in the diagnosis of peptic ulcer. The patients who were not x-rayed were diagnosed on the basis of the history and clinical observation.

At the Sixth General Hospital, where all patients were x-rayed, peptic ulcer constituted 8 per cent of the cases of chronic dyspepsia, as compared with 3 per cent at the gastrointestinal center of the Fifth Army. It would be expected that a higher percentage would be found at general hospitals, because they receive patients already observed clinically, in whom the suspicion of ulcer is therefore greater. The low incidence at the gastrointestinal center, where nearly all the patients were infantrymen who had engaged in heavy combat, is strong evidence that combat does not cause peptic ulcer.

The 33 per cent incidence of peptic ulcer among patients with chronic epigastric distress encountered in North Africa can be explained by the fact that this hospital, owing to its position, received more ulcer patients than did any other at the time. The experience of other general hospitals in the theater, however, is similar to ours in that far more ulcer patients were seen in North Africa than in Italy. It is probable that many more cases of the disease were not disclosed by the screening processes carried out in the Zone of the Interior earlier in the war.

Because the functions of Army hospitals often differ and because the nature of the work of this hospital has changed from time to time, a comparison of statistics with those of other reports in the literature would not be contributory. It appears, however, that the figures from this hospital are roughly the same as those in other American and British reports[1, 4] as regards the incidence of gastroduodenal disorders as a whole in respect to total medical admissions. The British consistently report a far higher ratio of peptic ulcer to non-ulcer dyspepsia than is found in American reports. The reasons for this discrepancy are not clear. It is certainly true that chronic dyspepsia and peptic ulcer together form an important military medical problem, although in the Mediterranean Theater the former was much the more important of the two.

A study of chronic non-ulcer dyspepsia cases in this hospital has been made and reported elsewhere.[5] That study was primarily an investigation of psychiatric factors in etiology. It was found that in more than three fourths of the cases the cause was psychogenic; that is, definite psychogenic factors were present with no other organic explanation. In the remainder of the cases positive evidence of significant neurotic factors was lacking, although there was likewise no demonstrable organic cause. In cases with definite psychogenic factors the diagnostic term "psychogenic dyspepsia" was employed. Among a few of the remainder a later follow-up with subsequent study revealed that many of these cases were likewise psychogenic in origin. It was found that patients with psychogenic dyspepsia who had been long in hospitals could rarely be salvaged for further military service. It was further noted that the personality structure of a large majority of patients with chronic psychogenic dyspepsia was basically different from that of a large majority of patients with peptic ulcer.

Method of Study

The large number of patients with peptic ulcer observed in a relatively short time forbade prolonged study, but data concerning the onset of the disease, with the possible influence on its course of various factors in the Army career, were accurately ascertained. Thus, in every patient the following data were noted: age, rank and branch of service; length of service; the duration of symptoms from the patient's earliest recollection of persistent indigestion (this was considered to be the onset of the disease); whether symptoms were aggravated on entrance into the Army, provided the disease had begun prior to induction; the date of departure for overseas service; whether symptoms had become aggravated coincident with overseas service (a period of time from two weeks before embarkation to four weeks after debarkation was arbitrarily chosen as the time of greatest nervous tension that might influence the symptomatology); and whether the patient had been in combat and, if so, whether he had been wounded. In addition to these data, it was noted whether the symptoms were typical or atypical of peptic ulcer and whether there was a family history of ulcer or chronic indigestion.

In 100 patients, a detailed history was taken, attention being paid to factors of emotional or neurotic significance. Thus, all phases of the past history were investigated, including the family history and the educational, occupational, social, marital and military phases of the patient's life. These patients were interviewed by one of us (H.W.), a psychologist on the Neuropsychiatric Service, to make a further personality study. Both of us then discussed the findings that each had made independently. An average of two hours' time was spent on the history and personality study of each patient. The purpose of this investigation was to evaluate the nervous and emotional factors that might be present in the personality pattern of the ulcer patient and to compare them with the findings made in 100 patients with chronic non-ulcer dyspepsia who were being studied at the same time in a similar manner.

The laboratory studies included stool examinations and blood-cell counts when indicated, and occasionally gastric analysis. The latter test was considered to be of relatively little value in the diagnosis, treatment or prognosis of peptic ulcer, except in cases with gastric lesions, in which x-ray examination alone does not always enable one to differentiate ulcer and carcinoma and in which the presence or absence of free hydrochloric acid is significant. This viewpoint is in agreement with that of Schindler[6] and Alvarez[7] (but not with that of Chamberlin[8]), each of whom has recently discussed the value of the procedure in Army gastroenterology. The test was performed only in patients with gastric ulcer, in the few patients who did not readily respond to treatment and in 3 patients with duodenal ulcer in whom anacidity had been reported from other hospitals. Free acid was found in the last group when histamine was employed as a stimulant.

As previously noted, it was our policy to make x-ray studies on all patients with chronic epigastric distress. A few patients who had come from other general hospitals and whose records and clinical findings indicated entirely satisfactory evidence of an active ulcer were not again x-rayed. No patients were included in the series who did not have a constant deformity of the duodenum or a crater. In many questionable cases the examination was repeated two or three times. Spot films with compression technic were taken in nearly all cases.

Clinical Aspects

Clinical Findings

In the taking of histories of patients with chronic

dyspepsia, the mental reactions of the vast majority of the patients with peptic ulcer were strikingly different from those of the non-ulcer patients. Most of the latter were anxious to describe their symptoms in detail, whereas the ulcer patients were noncommittal and unconcerned with themselves, other than for the single definite symptom —pain. In other words, the symptomatology of the patient with psychogenic dyspepsia was a direct expression of his anxiety and insecurity, whereas that of the patient with ulcer had no direct connection with anxiety. In making ward rounds, it was noted that the ulcer patients rarely complained or volunteered information regarding the degree of pain unless direct questions were put. From these observations alone it has been possible to predict with a considerable degree of accuracy what x-ray examination will reveal in patients with chronic epigastric distress, regardless of whether the symptoms were typical or atypical of ulcer. In most cases it could be predicted that the x-ray film would be negative in those patients who were anxious and unduly concerned with their stomach symptoms. Only rarely was an ulcer diagnosed by x-ray when the clinical impression had been that the patient did not have an ulcer. Much oftener a clinical impression of ulcer was not substantiated by x-ray. This lack of anxiety in the ulcer patients is in contrast to the findings of Flood,[9] who reports that 25 of 47 ulcer patients at the Station Hospital, Fort Meade, who had been studied neuropsychiatrically presented signs of anxiety. Schildkrout[10] reports 23 per cent of 87 cases of duodenal ulcer studied at a staging area in the United States, as having a psychoneurosis.

The symptomatology of the patients in this series was considered typical of ulcer in 72 per cent of the cases; that is, there was a characteristic indefinable, dull pain in the epigastrium, coming on one or two hours after meals and often in the middle of the night, which was relieved by food. Vomiting was an infrequent symptom. The symptomatology of the non-ulcer patients, although often resembling that of the ulcer patients with atypical symptoms, was rarely typical of ulcer. In patients who had had an ulcer for but a short time, the symptoms as related by the patient were often atypical — much more frequently so than in those who had had the disease for a long time.

The family history was ascertained in 167 patients. In 74 (44 per cent) some member of the immediate family had had chronic digestive difficulties. A similarly high incidence of familial predisposition to chronic indigestion has been noted in all studies of peptic ulcer. Whether this tendency is the result of a constitutional predisposition or carries a psychologic implication is uncertain.

The physical examination was negative in the vast majority of cases except for moderate epigastric tenderness, which was present in most cases. The x-ray examinations demonstrated a crater in 48 per cent of the cases so examined.

We were interested in the therapeutic response because of the lack of ideal facilities for treatment. Careful data could not be collected because the hospital stays before evacuation by transport to the Zone of the Interior were too short, being as a rule not more than two weeks. In the majority of cases, however, the degree of pain and discomfort was promptly lessened, usually within two or three days, and from then on the patients were quite comfortable.

The treatment instituted was the same in nearly all cases. Six-meal bland diets were provided, tincture of belladonna and alkaline powders being given as needed. The diet was limited in variety and not particularly palatable, owing to problems of supply. Feedings between meals consisted of powdered or evaporated milk, with or without crackers. Nearly all the patients were ambulatory. On admission, only 2 patients required complete bed rest with hourly feedings of milk. A few more were given for a few days two-hourly feedings of milk, with cereal, custard and crackers for the main meals. Tobacco was not prohibited. By and large, the symptoms of the entire series of patients were mild, responding promtply to the relatively crude dietary measures available. This finding is in contrast to Flood's[9] observation that the patients studied at the Station Hospital, Fort Meade, responded poorly, both symptomatically and roentgenologically. Our patients responded to treatment much as one would expect in such cases in civilian practice. Flood considered that the factor of anxiety, occasioned or intensified by the rigidity of Army life and by uncertainty for the future, was responsible for the poor response to treatment in his series, which was chiefly composed of recent inductees. The 11 patients who had had long Army service, on the other hand, responded well to treatment except for 2 who were highly neurotic. The patients in our series had had full military training and had come to a theater of operations. As soon as the diagnosis had been established they were told that they were to be sent home, so that the anxiety factor was largely removed. Thus, the good response to treatment when the anxiety factor was minimal, in contrast to the poor response in a different series in which this factor was relatively strong, is confirmatory of the prevailing belief that psychogenic factors are of importance in peptic ulcer.

In contrast to the almost universally good symptomatic response of patients with peptic ulcer to dietary treatment and hospital care, patients with psychogenic dyspepsia rarely improved symptomatically as a result of these measures. This observation provides a valuable clinical test in differential diagnosis.

Type of Ulcer

There were 188 cases of duodenal ulcer (94 per cent) and 12 cases of gastric ulcer (6 per cent), the latter incidence being essentially the same as that reported by Flood[9] and Annis[11] from two station hospitals in the United States. In the 12 cases of gastric ulcer (Table 1), the lesion was on the lesser curvature in 6 cases, in the prepyloric area in 5 — in 3 of which it was confirmed by operation — and in the antrum in 1 case. In 9 of the gastric ulcer cases the ulcer occurred after arrival overseas. These cases represented 18 per cent of the 50 cases in the entire series in which peptic ulcer developed after arrival overseas, as compared with 2 per cent of those in which disease had existed before going overseas.

TABLE 1. *Data on Cases of Gastric Ulcer (12 Cases).*

Case No.	Age	Race	Location	Duration	Remarks
	yr.				
1	45	White	Lesser curvature	5 mo.	
2	26	White	Prepyloric area	1 mo.	Perforation
3	33	Negro	Prepyloric area	2 yr.	Gastric resection, possible malignancy
4	37	Negro	Lesser curvature	2 mo.	
5	45	White	Lesser curvature	3 mo.	
6	34	Negro	Prepyloric area	4 mo.	
7	37	White	Antrum	8 mo.	
8	24	Negro	Lesser curvature	1½ mo.	
9	37	White	Lesser curvature	11 mo.	
10	26	White	Prepyloric area	7 mo.	Perforation
11	30	White	Prepyloric area	3 mo.	
12	24	Negro	Lesser curvature	5 days	Subacute perforation (no operation)

Racial Distribution

There were 16 Negroes with peptic ulcer, or 8 per cent of the series. This is probably a normal incidence, taking into account the small proportion of Negro troops in this theater. It does not substantiate statements that have been made to the effect that Negroes are less prone than Whites to develop this disease. In 10 of the 16 Negroes (62 per cent), the onset occurred after arrival overseas. There were 5 cases of gastric ulcer among Negroes, or 42 per cent of the number of these ulcers in the series, a surprising and inexplicable percentage, considering that only 8 per cent of the 200 patients in the series were Negroes.

Age, Rank and Branch of Service

The ages of the patients were neither greater nor less than the average for the Army as a whole. One hundred and seventeen patients (59 per cent) had received at least one promotion. Seventy-two per cent of the patients belonged to combat branches of the Army. Fifty-four patients had engaged in a slight to moderate amount of combat, and 9 had been wounded, none severely.

Complications

As noted previously, all the patients in this series had been sent to this hospital from other hospitals, where they had been treated initially. Because of this, there was only 1 case of major hemorrhage and 1 of subacute perforation. In the former case, the bleeding had begun on a transport from which the patient was transferred directly to this hospital, still bleeding moderately. A history of massive hemorrhage since arrival in this theater was obtained in 7 cases and one of perforation in 9 cases, the latter having been the first sign of the disease in 3 cases. Three of the cases of perforation were those of patients with gastric ulcer, and in 2 of these the perforation was in the prepyloric region, as verified at operation. The third patient, a Negro with an ulcer in the lesser curvature, was thought to have had a subacute perforation, since he had had severe pain at onset, with low fever and leukocytosis persisting for three weeks. He was not operated on.

It has been noted that the incidence of perforation in the civilian population of London during the two months of heaviest air raids in September and October, 1940, was three times as great as the normal average.[12] It has been thought that massive hemorrhage is especially frequent during periods of extreme nervous strain. The incidence of perforation and that of hemorrhage in this series — 4.5 and 3.5 per cent respectively — are not high, being essentially the same as those reported by Thomas[13] among 75 cases from Fort Meade. If psychogenic factors are important as precipitating causes for these complications, as suggested by Stewart and Winser,[12] the low incidence in this series may be due to the fact that the military population was composed of trained, disciplined soldiers.

Pyloric obstruction occurred in 3 patients, 2 of whom required gastric resection before they could be sent home.

Onset of Disease in Relation to Army Service

Reports in the literature on soldiers with ulcer studied in the United States agree that in 90 per cent the ulcers occurred prior to entry into the Army.[9, 14] This is in accordance with British figures.[15] According to Chamberlin,[14] these patients reach the hospital on an average of five months after induction. Ninety-three per cent of Flood's patients, who were recent inductees, had had symptoms prior to induction. Essentially all this group were discharged from the Army. The results of our inquiry into the onset of the disease in relation to time of entry into the Army are quite different.

In 113 patients (57 per cent) there was a history of unexplained indigestion or symptoms resembling those of peptic ulcer prior to entry into the Army. In 33 of these patients, a recurrence or aggravation of symptoms occurred within four weeks after entry into the Army, but not many of them sought medical attention. In 79 cases, recurrence or aggravation occurred shortly after arrival overseas. A few of

these patients were promptly hospitalized and sent home, but the majority paid no attention to the symptoms, remaining on duty until the symptoms had become extremely severe and often engaging in combat without reporting to sick call. In 87 cases (44 per cent), the disease began after entry into the Army. In 50 of these (25 per cent), it began after the patient had started for or arrived in the Theater of Operations. Twenty-four of the 37 patients whose ulcers had begun after entry into the Army but before coming overseas reported that their symptoms became much worse shortly after starting for overseas service. Thus, of 150 patients who had peptic ulcer before going overseas, in 103 (69 per cent) an aggravation of symptoms or recurrence of the ulcer could be directly correlated with nervous tension.

The 150 patients who had peptic ulcer prior to embarkation should theoretically have been excluded from overseas service. The reason why they were not may lie in the fact that most ulcer patients observed in overseas areas are restless and aggressive, and frequently conceal or minimize symptoms in order to be accepted in the Army or to be sent overseas. Berk and Frediani[16] state that their patients, who had not been overseas, did not conform to this type, that many were of low intelligence and that many made use of their ulcers to secure discharge from the Army. Thus, it may be that the ulcer patient who gets overseas is a different type of person in respect to aggressiveness.

Almost every patient's explanation of the recurrence or aggravation of his symptoms in the Theater of Operations was the difficulty of digesting the field rations on which he was obliged to subsist. That the accuracy of this is debatable is shown by the fact that the symptoms of many of these patients began or became aggravated at the port of embarkation or on the transport, and that when this occurred in the Theater of Operations it was not until they were subjected to the strain of air raids for the first time or started for the front, having been in the combat area and having eaten field rations for some time. This suggests that the main reason for the recurrence or aggravation of symptoms of peptic ulcer in these patients is not the type of food eaten but the nervous tension involved in overseas service.

Disposition of Patients

The patient with peptic ulcer as seen in the Theater of Operations is a highly successful soldier who as a rule has had his symptoms for a considerable period of time without complaining to a medical officer. The disease is clinically mild, and there have been relatively few complications. These patients have been of much greater value to the Army than the dyspeptic patients without organic disease. Yet it has been the practice promptly to send home practically every ulcer patient, whereas patients with non-ulcer dyspepsia are usually returned to duty or limited service, even though their disorder is generally more disabling and less amenable to treatment than peptic ulcer.

Patients with peptic ulcer in a theater of operations should in the large majority of cases be sent to the Zone of the Interior, because the chances of recurrence are extremely great and management of the disease outside a hospital is almost impossible. In this series, however, there were 6 patients in base organizations whose symptoms disappeared promptly and who were anxious to be returned to duty. After four to six weeks of hospitalization this was done. In view of the mildness of the disease as seen in this theater, the high military quality of most of the patients and the fact that in the United States nervous tension is lower and dietary conditions are better, it seems probable that many of the ulcer patients sent to the Zone of the Interior could be successfully utilized in a noncombat status within the United States after a few weeks of hospitalization.

Psychosomatic Features

In the foregoing sections certain statements regarding psychosomatic features have been made, inasmuch as they had an important bearing on the clinical aspects of the disease. These may be summarized as follows.

The extremely low incidence (3.4 per cent) of peptic ulcer among combat infantrymen hospitalized because of chronic dyspepsia is strong evidence that combat is not a factor in the cause of this disease. The reaction of patients with ulcer differed markedly from those with non-ulcer dyspepsia in that the latter were emotionally concerned with their stomach symptoms. Patients with a neurotic reaction rarely proved to have ulcer, even though the symptomatology resembled that of ulcer. Patients with peptic ulcer responded rapidly and satisfactorily to a crude dietary regimen. This differed from the response to treatment of a group in a station hospital in the Zone of the Interior, which may be interpreted as having been due to the patients' security in the knowledge that they were to be sent home. Lastly, there was a close correlation between aggravation of symptoms in a presumably active ulcer or recurrence of activity in a previously quiescent one, and the nervous tension associated with going overseas. Such a correlation was present in 103 of 150 patients.

It is believed by many workers that peptic ulcer is a psychosomatic disease in the sense that emotional disturbance with chronic nervous tension is an important factor, if not the primary one, in the etiology of the disease. No conclusive evidence can be drawn from the statistical data of this report in support of such a contention. Nearly 45 per cent of these patients developed ulcer after entry into the Army, and 25 per cent did so after coming

overseas. This is in contrast to reports from Army hospitals in the United States, where 90 to 95 per cent of patients with ulcer had had the disease prior to entry into the Army, and suggests that the nervous tension of Army life is at least a contributory cause of the disease. This difference may, however, be explained otherwise. The incidence of the disease is normally highest in men of military age. With the screening out of a large number of men with old ulcers in the United States before their units were sent overseas, the percentage of new ulcers that would normally be expected to develop in this age group should be higher in men who have been sent overseas because they have been longer in the Army.

Subsequent information obtained at the end of the war in Europe revealed that the incidence of peptic ulcer in the entire Mediterranean Theater of Operations was low, and probably lower than the expected incidence for men of military age.

New peptic ulcers were rare among infantrymen exposed in Italy to part of the most intense and difficult combat of the war. This indicates that fear, with the associated nervous tension, is not a factor in the etiology. New ulcers were seen more frequently among base troops than among combat troops, and this suggests that the frustration of regimentation with frequent periods of inactivity may be more damaging than actual danger, in which there are opportunities for release of energy through aggressive action.

It was to study the psychiatric features of patients with peptic ulcer that the personality study of 100 patients, described above, was made. From a psychiatric viewpoint, the study was a superficial one, since many hours with each patient would have been required to make an accurate and deep survey. Nevertheless, as a result of both the interviews and the observations of the patient's reactions made on daily ward rounds, it was possible to determine whether the patient was neurotic, to obtain data regarding his past environment and to get a clear picture, at least on a superficial level, of his characteristic personality pattern. This could be contrasted with that of the patient with psychogenic dyspepsia. The marked differences noted have proved to be of practical value in differential diagnosis. Many patients with psychogenic dyspepsia have no apparent anxiety, their only complaint being epigastric distress. In the Army, hospitalization of such patients is harmful, inasmuch as symptoms grow more fixed the longer they are studied and treated from a medical rather than a psychiatric standpoint. Early diagnosis and disposition from medical channels at forward hospitals, in many of which x-ray facilities are not available, has been shown to be of marked military value, as well as of definite therapeutic value, in such patients.[17]

Data regarding the background of the patients with peptic ulcer revealed two important features suggestive of emotional insecurity. Thirty-nine per cent of the patients had had an unfavorable childhood environment, such as separated or incompatible parents or severe economic distress resulting in neglect. Nineteen of 49 patients who had married were divorced or separated. Other factors that could be analyzed statistically were perhaps no more prominent than those to be found in any random group of men. Thus, 13 per cent of the patients had had enuresis in childhood, 20 per cent gave a history of anxiety phobias, such as fear of high places, of the sight of blood or of accidents, 20 per cent drank somewhat excessively on occasion, and 19 per cent had mild or moderate psychosexual conflicts.

Despite the presence of neurotic factors in a considerable proportion of the patients, only 5 per cent demonstrated a clinical neurosis. In other words, the large majority were not disabled by whatever neurotic traits were present. The patients with neuroses were abnormally concerned with their gastric symptoms, which seemed to have emotional value for them, and also showed evidence of anxiety. More extensive investigation would perhaps have disclosed in a larger number of patients significant neurotic features of an aggressive type with overcompensation. This has been demonstrated by Mittelmann, Wolff and Scharf.[18]

It has often been stated that the patient with peptic ulcer tends to be an unrelaxed, hard-driving, overaggressive person. We have noted this to be true as a general trend among our patients. In spite of a surface appearance of self-sufficiency, the characteristic ulcer patient, as we have observed him, is not relaxed and is not free of anxiety, as has been noted by other writers. He is not normal in his behavior and feelings, although his abnormal temperament leads to material success. This generalization is based on the fact that most of these patients when asked if they often felt nervous complained of being restless and unable to relax. The behavior on the ward of the large majority of patients was one of restlessness and impatience, thus confirming this statement. The ulcer patient, however, seems to solve his underlying insecurity differently from the patient with psychogenic dyspepsia. In contrast to the latter, he has developed an abnormal drive combined with restlessness and impatience by which he controls his anxiety. The dyspeptic patient magnifies his every illness and is overcome by it, whereas the ulcer patient shows an apparent indifference to the whole affair. Our ulcer patients conformed to the description given by Mittelmann, Wolff and Scharf,[18] whose patients showed assertive independence and self-sufficiency, covering underlying anxiety and insecurity and accompanied by feelings of resentment and hostility.

These differences between the two groups, although gained from a superficial personality study

have been so striking as to leave no doubt that they actually exist. The main personality features that have been described were surprisingly uniform and definite. In the regimented life of the Army, when all men live exactly alike, exposed to the same difficulties and the same needs for adjustment, and where the opportunities for compromise existing in civilian life are lacking, the differences are doubtless far more obvious than they would be in civilian practice. Additional studies of patients of the latter sort, both men and women, are needed to confirm or disprove the conclusion drawn from this study of men living under regimented conditions, to the effect that those who develop peptic ulcers have a personality pattern fundamentally different from that of those who develop psychogenic dyspepsia.

If one accepts the hypothesis that psychogenic factors are important in the etiology of peptic ulcer, it is illogical to assume that psychogenic dyspepsia, in which the personality type differs markedly from that in peptic ulcer, should develop into the latter. Schildkrout[10] and Urquhart[19] have commented on the low incidence of functional dyspepsias that later develop ulcer, the latter finding it to be less than 3 per cent. If such did occur, one would expect combat infantrymen to develop the disease in large numbers, inasmuch as psychogenic dyspepsia is extremely frequent among them,[17] yet it is extremely rare.

On the other hand, that peptic ulcer is a psychosomatic disease is not yet proved. The above data, both statistical and psychiatric, are consistent with such a theory, yet add no conclusive supporting evidence. The discrepancy between our findings and those of Flood,[9] Berk and Frediani[16] and Schildkrout[10] on soldiers with ulcer in the United States as regards personality factors and response to treatment has several possible explanations, among which are the following. Standards of psychiatric evaluation, always largely subjective, may have differed. Environmental factors are known to affect the form of any psychologic reaction. For example, severely wounded soldiers react quite differently to pain from civilians with comparable injuries. Finally, it may be that the man with pre-existing ulcer who manages to get overseas is actually a more aggressive, effective soldier than those who remain in the United States.

It is hoped that the observations reported herein may be of use in integrating the many factors involved in the psychosomatic aspects of peptic ulcer. Much further work will be required before a psychogenic factor can be assigned a specific major role in the etiology of this disease.

Summary and Conclusions

A clinical study has been made of 200 consecutive cases of peptic ulcer seen in a general hospital in the Mediterranean Theater of Operations. The disease was mild, and the response to treatment was prompt and satisfactory, being similar to or more favorable than that observed in civilian practice. The incidence of major complications was no higher than that noted among soldiers in the United States.

A study of psychosomatic features revealed that distinct episodes of nervous stress could be correlated with an aggravation of symptoms or recurrence of previously existing ulcer in 69 per cent of 150 patients whose disease had begun before coming overseas. Only 3.4 per cent of 183 combat infantrymen with chronic epigastric distress had peptic ulcer. The disease was more frequent among base troops than among combat troops.

Forty-four per cent of the patients developed the disease after entry into the Army, and 25 per cent developed it after beginning overseas duty. Reasons are given for believing that these figures are not significant as indications that the nervous tension of Army life is a direct factor in etiology of new ulcers.

The results of a psychiatric study of 100 patients were consistent with those of previously reported work on the personality pattern of patients with peptic ulcer. This pattern was markedly different from that of the large majority of patients with psychogenic dyspepsia, and is valuable as a differential point between the two disorders.

Reasons are given for believing that psychogenic dyspepsia does not evolve into peptic ulcer.

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THE RESULTS OF SECONDARY CLOSURE

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IT HAS been observed that the victory in World War II is likely to be credited to penicillin and secondary closures. The humor of this remark detracts in no way from the importance attached to these surgical aids by those who have witnessed their proper application.

During the last nine months we have done secondary closures on 1450 patients, these closures constituting 43 per cent of all operative procedures. Since many of these patients had multiple wounds, as many as thirty wounds having been closed in a single case, the total number of wounds closed is well over two thousand. It would be meaningless to give over-all percentages of results regardless of the antecedent histories, since this would combine the favorable cases having a normal expectancy with those presenting little hope of success, and thus give a true picture of neither group. Because of the vicissitudes of the process of evacuation, patients have arrived with every conceivable type of wounds — early and late ones; those not touched since their débridement and those frequently dressed; wounds with accompanying trauma to blood vessels, nerves, tendons and bone; and wounds of all sizes and shapes and in all locations. As regards cases with extensive loss of tissue and exposure of important structures, the choice of the appropriate plastic procedure requires discussion in specialized articles, but the ordinary flesh wound needs primary consideration because the basic principles of its treatment apply in almost all war injuries.

Our introduction to battle casualties gave us a mixed group of cases many of which were too old for successful closure. These received wet dressings and many of them were finally closed, but we seldom obtained better results than an indurated scar having wide suture marks and causing difficulty in mobilizing the adjacent joints. It was obvious that once a wound became infected the end result would be impaired even though the wound were properly cleaned before closure. It was equally obvious that open wounds would become infected even with careful dressings. The logical answer seemed to be to close the wound at the *first* dressing after débridement. It has been our policy to do this whenever possible, and experience indicates that it gives the best results.

After the first convoys mentioned above had arrived, we received patients evacuated by air, many of whom had wounds that were only two or three days old. These cases were scheduled for operation without even preliminary ward inspection. This policy caused a few "dry runs," but it was believed that the advantage gained in the large majority of cases warranted this inconvenience. There were occasional small wounds that did not require closure — cases with perforating small-arms wounds in which it was not desirable to seal up a long tract and with odd septic wounds. Cases with a favorable history, that is, one of adequate and prompt débridement and no intervening dressings, amounted to no more than 5 to 8 per cent of the total.

As the battle front got farther away, cases as old as three or four weeks again began to arrive. These had usually been dressed many times, and the incidence of infection was so high that it was no longer permissible to close them immediately, and it was necessary to revert to preliminary wet dressings, with a corresponding worsening of end results. The evacuation pendulum finally again swung in our favor, and recent convoys have brought in patients with wounds four, five and six days old. Once more the operating rooms were kept going at full speed so that all these wounds could be closed at the first dressing. The results were satisfactory.

This varying experience has shown that the results of secondary closures can be quite accurately predicted on the basis of the conditions under which the operation is performed. With a prompt and adequate débridement and no intervening dressings, wounds that are closed by the end of the first week have healed without complications in 95 per cent of cases. In less than 2 per cent of such cases frank sepsis has been encountered. Only 3 per cent have developed hematomas that had to be evacuated. Moreover, these wounds heal with a minimum of induration and inflammatory reaction. After ten days, as the number of intervening dressings and the time interval between them increase, there is a corresponding decrease in the percentage of wounds that can be closed at the first dressing, reaching zero in about three weeks. In the cases of this group and those that are closed after being cleaned, a percentage of satisfactory results of 60 per cent is the highest that can be expected. Here one encounters much more drainage, breaking down of all or a portion of the wound and considerable inflammatory reaction, cutting in of sutures and finally greater scar contracture.

Débridement

The value of prompt and complete débridement is easily seen at the time of attempted closure. The pressure of action at the front is quickly re-

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flected in this initial surgery. We have been impressed by the speed with which these wounds first come to débridement and by the excellent work done by the evacuation hospitals. Sometimes, of course, through force of circumstances, delay is unavoidable and débridement is incompletely done. This is always apparent in the condition of the wound and necessitates the placing of preliminary wet dressings before closure can be attempted. Bits of foreign material have sometimes been found in such wounds when it was obvious that the pressure of casualties had allowed less careful treatment. The desirability of adequate débridement needs no emphasis to the tireless workers who have done such perfect work in the advance units. Testimony of the effectiveness of their work is furnished by the fact that a large percentage of wounds reached us in such perfect condition as to permit immediate closure.

Intermediate Dressings

Most patients consider that a surgical dressing is evidence of careful medical attention. That wounds should be débrided and dressed seems reasonable to the layman. A white gauze bandage quickly becomes soiled, is frequently loosened and is usually blood stained. To remove this unsightly dressing and apply a clean, firm, fresh one pleases most patients and too many physicians. That every change of dressing, even under the most favorable conditions and with the best aseptic technic, means introduction of infection is a fact not sufficiently recognized, and when the conditions are not so ideal and the technic not so scrupulous, the danger is considerably increased. One needs only to observe the contrast in the clinical appearance of wounds coming to a general hospital to appreciate this fact. The ideal wound for closure is the one which has its débridement dressing removed in the operating room just before suture. Most wounds associated with fractures and therefore encased in plaster casts, which effectively discourage intermediate dressings, are cleaner than simple flesh injuries, even though they are generally much more extensive. Perhaps all débrided wounds should be dressed with a light plaster, which would afford greater protection to the damaged tissues and be more resistant to soiling and disarrangement, and thus less likely to invite a change of dressing. Certainly intermediate dressings should be discouraged unless there is a definite indication for them, such as fresh bleeding or systemic evidence of infection.

Chemotherapy

All patients received through American installations had already received chemotherapy in some form. Penicillin has been given routinely for forty-eight hours after closure, but not after that time except for specific indications. The excellent condition in which the ideally treated wound is found is considered in some measure due to previous chemotherapy. Certainly when one sees extensive abdominal wounds and patients with multiple compound fractures, the wonder frequently is that they have survived long enough to reach a general hospital. There can be little doubt that many of these patients owe their lives to early chemotherapy. Once they have reached this hospital, an adequate defense mechanism has been established, and only enough penicillin is needed to tide them over whatever breakdown of the walling-off process is incident to the secondary closure.

Method of Closure

As indicated above, we prefer to book candidates for secondary closure without prior inspection and to have the dressing initially removed on the operating table. On removal of the vaseline gauze with which the wound should be loosely packed, the tissue should be beefy red, with no foreign material and no suppuration or slough. The skin borders should be pliable and elastic, with no surrounding induration or inflammatory reaction. Usually the general appearance of the wound invites closure. Some wounds have a mild grayish exudate with a slightly glistening sheen, which has not been found to be a danger signal. We do not apply antiseptics to the area or even wash out the wound itself. The wound is covered with a sterile sponge while the surrounding skin area is being prepared. In most cases preparation consists of painting the wound with two coats of Tincture of Merthiolate and one of alcohol. If the dressing has been disarranged and the area around the wound thus contaminated, it is cleaned with a sponge moistened with tincture of green soap or ether before applying the antiseptic solutions. It is thought that too vigorous scrubbing of the area may wash bacteria into the wound, so that the skin is washed at this time only when such a step is visibly indicated. The wound itself is not prepared in any way. The presence of a little vaseline does not interfere with the healing of closures or with grafting procedures.

It takes some experience to judge whether a wound is of suitable size and shape for direct closure. With more experience, it will probably be possible to apply this procedure to seemingly hopeless cases. It has been found that even considerable tension is tolerated if other conditions are favorable. Looseness of the skin is a distinct advantage. Wounds of the back, chest, abdomen and thighs are more easily closed than are much smaller ones on the forearm or lower leg. Special caution has been observed in dealing with wounds of the buttocks, and in many cases it has been considered best to strap them with fenestrated adhesive strips. This method in fact produces almost as good a closure as does suturing, and it is preferable when there is any possibility of sepsis. It has the advantage of

not sealing the wound and of pushing rather than pulling its edges together. It gives better results with deep wounds than with shallow ones, in which it has a tendency to roll the edges inward. An extensive wound that laid the buttock open down to the sciatic notch and extended from the sacrum to the thigh was closed with wide adhesive "butterfly" straps, with excellent healing and a completely satisfactory scar.

Closure has been accomplished largely with deep interrupted silk sutures, the bottom of the wound being picked up to obliterate dead space. Only rarely have buried sutures been used. If it is considered necessary to close the fascia of the thigh with buried catgut, a Penrose drain is inserted under the fascia and carried out through the most dependent portion. The drain is removed in forty-eight hours. If necessary, the skin margins are unhesitatingly freed to facilitate approximation.

If the size, shape or location of the wound prohibits direct closure, some type of grafting is indicated. The indications and technic for these procedures require special consideration and will not be discussed herein, but the principle of early closure at the time of the first dressing applies equally well to plastic procedures. In other words, the wound should be closed at the optimum time by whatever method is necessary — simple closure if feasible, a split-thickness graft if the difficulty is just a question of too much skin loss, or a pedicle graft for gouged-out wounds exposing vital structures. In some wounds a combination of all three methods is required.

* * *

Whatever the method of closure, it never has so favorable a chance of success as when done at the first dressing by the end of the first week. Complete epithelialization at this time affords the best possible dressing, lessens the incidence of infection, obviates painful dressings, conserves material and reduces nursing care and, most important, improves the chances for a good functional result. The goal of the evacuation system should be to get these patients to a hospital in which their wounds can be closed by the end of the first week. Intermediate dressings en route should be discouraged unless they are absolutely necessary.

MEDICAL EDUCATION AND PRACTICE IN GERMANY DURING THE WAR

Colonel Robert M. Zollinger, M.C., A.U.S.

INFORMATION concerning medical education and practice in Germany has been scanty during World War II. Because of the past achievements of German medicine it was considered important, both for the present and for the future, to attempt to evaluate its status during the last five secretive years. Certainly the standards of medical and surgical care of the German soldier at the front were not impressive. Only after the defeat of his country was it possible to visit the medical centers responsible for the education of the German medical profession. The following recent impressions were gained after visiting the clinics of the professors of medicine and surgery in three prominent German medical schools.

Medical Education

Source of Students

The quality of any professional school is founded not only on the educational and research facilities offered but also on the caliber of the students themselves. All the professors of the medical schools visited agreed that the caliber of the students was far below the prewar level. They recognized this as an alarming situation and believed that it was due to the following factors.

The majority of the students came directly from the Army, and in many instances they had been actuated only by a desire to avoid the hardships and dangers of combat. Consequently, they had expressed a desire to study medicine and had therefore been released by the Army for such study. In most cases they had neither the background nor the aptitude for study in a medical school. The students in this group consisted of physically fit men who were part of the so-called "Student Combatant Corps." They were under the direction of an Army officer in each school. They wore uniforms and were committed to the Army while at school and at the end of their studies. Their tuition was apparently paid by the State.

A smaller group consisted of soldiers discharged from the services because of general physical disability or for various other reasons. The State paid for their education and directed their assignment to a civilian or static military hospital after the completion of their education.

Another group consisted of those initially unfit for military service and of a few who had been excused from it because they had demonstrated a particular aptitude for the study of medicine. These students paid their own tuition, which amounted to 300 to 400 marks yearly, and their living expenses, which approximated 120 marks monthly. After completion of their studies, they were assigned to civilian or static military hospitals. A select few were trained as specialists following graduation.

The last group consisted of an increasing number of women who were admitted to the universities to

study medicine. They were eventually assigned to civilian hospitals or trained as specialists. It was evident that the various professors did not desire women as their assistants or as students for prolonged training leading to eventual certification in a specialty.

Thus, the main difference in the selection of students, as compared with the prewar method, was that the majority were admitted by appointment from an outside authority. There was no apparent screening by an entrance board, and many students who had a natural aptitude and desire for the study of medicine never had the opportunity to enter medical school.

Undergraduate Training

Without question, the war drastically interfered with the training of medical students. In the first place, the total length of study — premedical and medical — was decreased from six years, or twelve semesters, to five years, or ten semesters. Finally, early in 1945 it was further reduced to four and a half years, or nine semesters. Since the majority of the students were under control of the Army, they had many interruptions associated with military training and Nazi Party functions. At least one semester of the premedical years was spent by the student as a medical corpsman at the front. During the three medical years he had to spend approximately two semesters working in an Army hospital at the front. Probably the students with considerable previous military service were excused from some of these assignments. There seemed to be no systematic arrangement for the assignment of these students to Army service for any given period of time. Apparently, groups were sent throughout the year for periods ranging from a few weeks to three months. Ordinarily, in the years of clinical study they would have worked for a period of three months in each of the various clinics, such as those of surgery, medicine and otolaryngology. As a result of this Army service, many must have missed the opportunity for bedside teaching and clinical work in hospitals other than that afforded at the front. Certainly, no faculty under the circumstances could have juggled a curriculum to cover the needs of such a fluctuating student body. The total time actually spent in the university, including the premedical and medical terms, was in reality limited in the majority of cases to about three and a half years.

The effect of the above-mentioned factors alone was sufficient to produce a drastic lowering in the quality of medical graduates. There were, however, other serious influences, which were brought out by all the professors. They emphasized the effects of the general hardships of a prolonged war and the attitude naturally associated with war as distracting even the better students from serious study and application. Education had been further interfered with, especially during the previous eighteen months, by air-raid alerts and the destruction of buildings by bombing, with consequent overcrowding in other buildings. For example, although lectures were begun at 8 a.m., by 10 a.m. there was usually an air-raid alert, which compelled students to enter shelters and remain there until noon. Frequently there was another alert at about 2 p.m.; evening study was interrupted by alerts, and sleep was constantly broken by alerts that came at about 2:00 a.m. As a result of aerial warfare, not only was study and sleep interfered with, but only about half the time was it possible to attend classes. As the result of bombing of buildings, one of the professors of surgery had as many as 250 students in one class. Since the number of assistants decreased by one half and since the civilian patient load was quite heavy, it was impossible for the teachers to instruct the students as thoroughly as had been possible in peacetime.

Postgraduate Training

In peacetime, the professors of medicine and surgery had approximately twenty to forty assistants, depending on the size of their clinics, but during the war the number was reduced by at least half. Assistants were appointed from a group of promising students who applied to the professor of their choice for postgraduate training in some specialty. If the applicant was an able-bodied man, he had to be deferred by the Army as nonessential and in addition had to be cleared by the Ministry of Education. The number of men available was so limited that it was necessary for the professors to accept an increasing number of women as assistants each year.

Assistants were paid from 5000 to 7000 marks yearly, depending on the length of service. At least one of them was used in the private clinic of the professor concerned for one year. Occasionally the professor would arrange for the assistant to increase his income by caring for semiprivate patients in the clinic and by filling out insurance reports and other papers. Health insurance appeared to be quite popular with the teaching staff.

In surgery, at least, a minimum of four years was required before the professor issued a certificate attesting that the assistant was qualified in his specialty. Assistants showing particular ability in teaching and research might be retained for as long as ten years. They were given more responsibility each year and were finally qualified for teaching appointments in the universities.

In peacetime and early in the war, at least two Army officers were sent to each clinic for further study in surgery and medicine. Before the end of the war the number of Army doctors sent and the time spent in the various clinics were variable. These men were assigned by the Army, and the number and type of students were no longer controlled by the professors.

Research

It was evident that there was little opportunity for research, and as a result few significant contributions in this field were made during the war. Although the various professors appeared to be of high caliber, and had in the past carried out or supervised extensive research projects, this activity during the war was greatly curtailed by many factors. In the first place, as already mentioned, the number of assistants was decreased by half. The students were of such poor quality that few had either the time or the inclination to assist in research projects. In some instances the professors had served for long periods as consultants in the Army. On their return to civilian life they were burdened with a tremendous professional load of civilian patients and casualties from air raids. In some cases extensive damage had been done to the clinics and laboratories, which further impeded research. In some clinics all the surgery had been performed during the past eighteen months in bombproof, partially underground hospitals of limited space. There was also an understandable shortage of drugs and supplies for patients, let alone research projects. The majority of professors, however, attempted to continue their investigations pertaining to subjects that had interested them for years, but many times they were unable to publish their findings or to have reprints of their articles made because of the paper shortage, or because of arbitrary discriminations of one kind or another made by Nazi officials. Apparently the latter were especially active in curtailing the publication of articles pertaining to a medicohistorical subject.

It is fair to say that individually the professors expressed resentment of the occasional appointment by the Nazis of inferior men to head departments, although such interference was apparently limited to certain universities. Furthermore, they appreciated keenly the loss to the universities in the last few years of some men as a result of racial discrimination. Inquiry was made of each professor regarding what he considered to be the greatest advance made during the war; invariably the answer was, "The use of sulfonamides." Second in importance was the rather widespread use of whole-blood transfusion. Penicillin had never been made available.

Medical Practice

Surgery

Specific inquiry was made concerning the principles of therapy utilized for some of the more frequent general surgical conditions. The various specialties were not evaluated. Although the basic principles of good surgery appeared to be thoroughly understood, it was evident that many practices were years behind the times. Although the individual clinics may have standardized their procedures, there was little evidence given by the men who had served as senior consultants in the Army that a systematic routine for the management of battle casualties was practiced.

Sulfonamide therapy was considered the greatest advance of the war, and many different varieties of the drug, comparable to those available in the United States, were utilized. Although enthusiastic, the surgeons did not have so much confidence in the sulfonamides as did the internists. Certainly, in two of the large clinics they were not sure of the value of sulfonamides when applied locally in open wounds. Apparently both in civilian practice and in the Army it was optional with the surgeon whether sulfonamides were dusted into wounds. They all emphasized that adequate surgery was more important than chemotherapy. Sulfonamides were used in the treatment of gas gangrene, but the surgeons had no confidence in their effectiveness.

All wounds were débrided if seen within forty-eight hours after the time of injury, and the majority were left open. The value of early secondary closure for most wounds was not appreciated. A few large wounds were secondarily closed from five to seven days after débridement, but this was not a routine procedure in either civilian or Army practice.

Gas-gangrene antitoxin was used in the Army for wounds in susceptible areas, such as those of the upper thigh. There was general agreement that adequate radical surgery was the only efficient method of preventing gas gangrene, as well as of treating it after infection had developed. Little confidence was expressed in gas-gangrene antitoxin or the sulfonamide drugs in the management of such cases.

Relatively little tetanus was seen in civilian practice, although there had been a noted increase during the last six months. There was a tendency to attribute this to the fact that the recently manufactured antitoxin was probably defective. Tetanus antitoxin seems to have been given to all wounded men, and tetanus toxoid was reserved for special troops. Once tetanus had developed, it was treated by the daily injection of antitoxin, either intravenously or intrathecally. Avertin was used to control muscle spasm.

The treatment of burns appeared to be fairly standardized in the several clinics visited. In most of them the wound was débrided by brushing and by the application of alcohol under general anesthesia. Following this, a powder, whose active principle was tannin, was applied and the area was covered with dry gauze. In some cases boric acid and vaseline — and in others cod-liver oil — were applied to the burned areas. There was a tendency to give whole-blood transfusions daily; plasma was given only as a substitute. Pressure dressings, immobilization, early skin grafting and the requirements of blood and plasma replacement were not appreciated.

It was mentioned by a certain surgeon that one of the greatest advances in neurosurgery was the primary closure of compound wounds of the brain. Apparently primary suture of severed nerves at the time of injury was not practiced, but there was an attempt at loose approximation with one or more silk guide sutures. Secondary suture for injuries of the radial and ulnar nerves below the elbow was done four months after injury, that for nerve injuries above the elbow six months after injury, and that for division of the sciatic nerve eight months after injury. Early anastomosis of the nerve was not practiced, although it was generally agreed that better results might be obtained if early suture were attempted and if neurolysis were carried out more frequently.

The treatment of chest wounds was neither standardized nor modern in its concepts. Foreign bodies with rough edges larger than 1.5 cm. in diameter were sometimes removed. Smooth asymptomatic foreign bodies were not removed unless there was associated hemorrhage, abscess or pain. Hemothorax was treated by occasional aspirations during the first four weeks, but no attempt was made to keep the pleural spaces completely dry. Decortication of the lung was not generally practiced, and it is probable that the indications for this operation were not understood. Empyemas were treated with constant suction for approximately three weeks through a No. 16 catheter; irrigations were not utilized during this period.

So far as could be determined, there were no new principles governing abdominal surgery; colostomies were not routinely performed for injuries to the large bowel. Sulfonamides were not applied within the intraperitoneal cavity unless there was evidence of established infection; but under such circumstances there was no hesitation in introducing as much as 20 gm. of the drug.

Some attempts had been made to perform blood-vessel anastomosis, with fine silk as suture material. Heparin was apparently not available, and no special technics for anastomosis were developed. Occasionally the femoral vein was ligated as a prophylactic measure in the presence of pulmonary infarcts.

Extremely few early cases of trench foot had been treated in the civilian clinics visited. Those in charge were primarily concerned with the late complications, and invariably recommended paravertebral block of the lower two or three lumbar sympathetic ganglions, 10 cc. of 0.75 per cent novocain being used in each site. For troublesome causalgia, 1 cc. of 70 per cent alcohol was injected in the vicinity of the appropriate ganglion after the preliminary injection of novocain.

The surgeons used vitamin preparations, but did not have so much confidence in this form of therapy as did the internists. Information was volunteered that children with infections who had been treated in relatively dark bombproof hospitals for as long as six months and had got little sunshine seemed to do about as well as did those treated in modern hospitals where plenty of sunshine was available. Vitamin K did not seem to be used extensively, if at all. Patients with intense jaundice were treated with calcium and blood transfusions.

There was no evidence of the use of plasma or any type of amino-acid preparations for the intravenous therapy of malnutrition.

Although there was general agreement that the widespread use of whole-blood transfusion constituted one of the major advances of the war, there was little evidence that the actual requirements of whole blood in the various surgical conditions were understood. Transfusion was largely effected by an antiquated direct method, although occasionally the citrate method was employed; neither glucose nor refrigeration seemed to have been in use as a preservative in civilian hospitals. Each city had a list of donors, usually workers in large nearby factories, so that whole blood was readily available. The donors were paid 25 marks for each transfusion (500 cc.), and in addition were given extra coupons for food rations; these incentives were sufficient to ensure an adequate supply of donors at all times. Wet plasma was available, but neither the dosage of blood nor that of plasma appeared to be controlled by laboratory tests or to be based on previous scientific estimations of the requirements for any particular type of case. No information concerning the use of plasma substitutes was obtained.

The operating rooms in the various clinics did not contain a single modern anesthesia machine. It may be that some of these machines had been sent to the Army or stored outside the city for protection against bombing. It could not be determined, however, that the surgeons employed gas anesthesia, and they seem to have had little experience with intratracheal anesthesia. They used 0.5 to 1.0 per cent novocain for local anesthesia in many operations, and barbiturates were widely employed intravenously. Ether was used for inhalation anesthesia except in cases presenting pulmonary infection, in which chloroform was used. There was little mention of spinal anesthesia, which was certainly not used in surgery of the upper abdomen. Morphine, pantopon, dilaudid and scopolamine were utilized in various combinations.

No new instruments or modern devices were observed in any of the clinics visited. Some of the surgeons, however, were enthusiastic about the use of long metal rods that were introduced into the medullary canal in the treatment of fractures of the long bones, especially those of the femur. They did not employ scalpels with detachable blades. Sewing clamps of the von Petz type were available for gastric and intestinal surgery. In most clinics an electrocoagulation and a cutting unit were available. Casual observation tended to confirm the

fact that the various types of laboratory and x-ray equipment were of good quality, although sharply limited in quantity. In one of the clinics the technical steps of a gastric resection were observed, and these were consistent with the highest standards of surgical technic.

The bomb-proof hospitals were of considerable interest. They consisted of thick concrete-and-steel walls, which were supposedly bomb-proof. Most of them were partially underground and were connected with the main clinic by an overhead as well as an underground passage. The units were self-containing in that they had their own ventilating system, electric generating system and artesian wells for supplying water. The windows were mere slits, resembling those used by archers to defend the entrances of medieval castles and fortresses. Such units were occasionally large enough to protect at least 100 patients, women and children being given priority for bed space. Such cramped quarters certainly greatly limited teaching facilities.

Medicine

The various professors of medicine appeared to have carried out more research, or at least to have written more papers, than had the surgeons. They were enthusiastic about the sulfonamides and were eager to obtain penicillin for both experimental and therapeutic uses.

One of the problems had been the investigation of synthetic foods, especially butter derived from coal tar. By colorimetric methods it was possible to determine that about 90 per cent of this substance was absorbed by the body. The professors would not admit that the treatment of malnutrition had ever been a serious problem, and they depended on oral feedings for supplying protein deficiencies.

The treatment of typhus fever was apparently symptomatic, and the results obtained from anti-typhus serum were variable and not entirely satisfactory. The professors had gained the impression that the initial mortality from this disease among Germans and French was much higher than that among other Europeans — that is, Russians and Poles. Hepatitis was not infrequently encountered, especially among children, and many patients had been believed to have hepatitis without jaundice. The presence of jaundice-producing virus had been proved, and the virus had been transmitted from egg to egg fourteen consecutive times. The treatment of syphilis was limited to the giving of arsenicals, since penicillin was not available.

* * *

During the recent conflict little was known of the German medical profession, and many have thought that because of its past ingenuity and skill it might have made many valuable discoveries. It is now apparent that the myth of German superiority in the medical profession is as much a fallacy as it was found to be in other forms of German endeavor. This deterioration has been due in part to the curtailments resulting from a long war, but prolonged interference and regulation by the State in the selection and activities of the students, as well as of the teaching staffs, did irreparable damage to the German medical profession. Although political interference and state control have been carried to extremes in Germany, the tendency for such influences to stifle high professional standards in any country must not be overlooked.

That any significant new developments have been made in Germany, either in medicine or in surgery, in recent years is doubtful. The practices of therapy there are in most instances antiquated as judged by our standards, and it will unquestionably be years before the German medical profession regains, if it does so at all, the high position that it held twenty or thirty years ago.

CARBOHYDRATE METABOLISM IN NORMAL PREGNANCY

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BOSTON

GLYCOSURIA during pregnancy is such a frequent occurrence that it is often dismissed as unimportant. We have found, however, that true diabetes often begins during pregnancy and may be overlooked and that pre-existing diabetes is often made worse by pregnancy. These two facts prompted us to study the effect of normal pregnancy on carbohydrate metabolism.

METHOD

Twenty-five healthy pregnant patients were taken at random from the clinic of the Boston Lying-in Hospital. Glucose-tolerance tests were done in each trimester of the pregnancy, one or two weeks after delivery and several months post partum. The capillary blood-sugar level was determined fasting and half an hour, one hour, two hours and three hours after the ingestion of 1 gm. of glucose per kilogram of body weight. Urine sugar tests were made at the same time. The patients were kept on full normal diets, since the distorting effect of low-carbohydrate diets on glucose-tolerance curves is well known. The blood-sugar analyses were done by the method of Folin.[1] Complete data were obtained in 11 cases; those for the other 14 cases were complete except for one or two curves.

RESULTS

Figure 1 shows the blood-sugar curves for each trimester, immediately after delivery and several months post partum. Each curve shows the fasting value, the peak — usually the highest point in the curve but not always the one-hour value — and the two-hour and three-hour values.

The fasting blood-sugar levels during pregnancy were well within normal limits. Most of them were in the low-normal range, over half being under 90 mg. per 100 cc. Only 1 patient had a level of more than 120 mg., — 126 mg., — and subsequent levels in the same patient were below 95 mg., so that this high value was probably due to a technical error. The post-partum group showed somewhat higher fasting blood sugars, only 1 patient having one under 90 mg. per 100 cc.

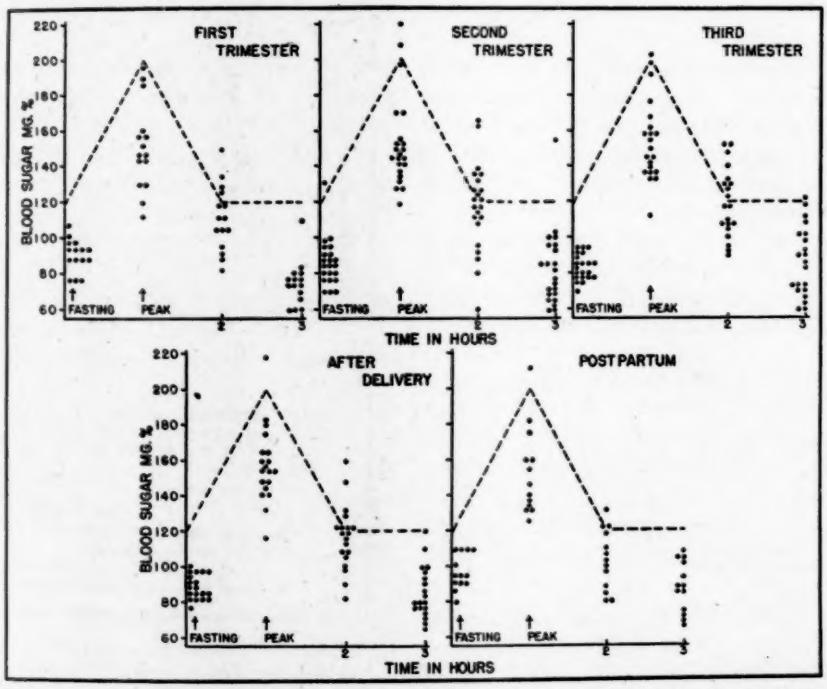

FIGURE 1. *Glucose-Tolerance Curves during Pregnancy (each trimester), after Delivery (one to two weeks) and Post Partum (three or more months).*

The broken lines in each chart represent the curve for normal young adults (peak, 200 mg. or less; two-hour level, 120 mg. or less; and three-hour level, 120 mg. or less).

The peaks, too, were within normal limits. In young patients, the peak when capillary blood is used may be as high as 200 mg. per 100 cc.,[2] since the rise is higher than that in venous blood. Here

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also the pregnancy values were with rare exceptions within normal limits.

The two-hour levels, however, showed a high percentage of abnormally high values during pregnancy. The capillary blood-sugar level should fall to normal — below 120 mg. per 100 cc. — in young persons.[2] It is apparent from Figure 1 that many high two-hour values are present during pregnancy and after delivery.

The percentages of abnormally high two-hour values are given in Table 1. The highest percentage of abnormal two-hour levels occurred in the second and third trimesters and after delivery. Occasional values were as high as 164 mg. per 100 cc. Only 2 patients in the post-partum group had high levels, and these were just over the critical level — 121 and 127 mg., respectively.

TABLE 1. *Percentage of Patients with High Two-Hour Blood-Sugar Levels.*

PERIOD	PERCENTAGE
First trimester	29
Second trimester	52
Third trimester	45
Sometime during pregnancy	81
Immediately after delivery	47
Postpartum	15

All the three-hour levels were normal throughout pregnancy and the post-partum period, except for two high values in 1 case. The values in this case are given in Table 2. There was an extremely high three-hour level — 155 mg. — in the first test. This followed a lower two-hour level, which suggests a technical error. The next curve gave a three-hour level of 122 mg., which is just over the normal value. Subsequent curves were normal.

TABLE 2. *Results of Glucose-Tolerance Tests in a Single Case.*

PERIOD	FASTING LEVEL	PEAK LEVEL	2-HOUR LEVEL	3-HOUR LEVEL
	mg./100 cc.	mg./100 cc.	mg./100 cc.	mg./100 cc.
Second trimester*	96	143	136	155
Third trimester	93	162	131	122
Immediately after delivery	88	161	147	110
Postpartum	90	160	97	105

*Patient first seen in second trimester.

DISCUSSION

The above data indicate that normal pregnancy exerts a deleterious effect on carbohydrate metabolism, as is shown by the distortion of the glucose-tolerance tests. Two-hour blood-sugar levels over 120 mg. were extremely frequent, especially during and after the second trimester. These persisted after delivery but returned to normal several months post partum. It is during the second trimester that diabetes often appears and that pre-existing diabetes is made worse.[3] Obviously, if a high two-hour blood-sugar level is considered diagnostic of diabetes, many normal pregnant patients — 81 per cent of this series—would be so diagnosed. On the other hand, all the three-hour levels during pregnancy were normal except for those in 1 patient, in whom subsequent determinations during pregnancy were normal. It is apparent that diabetes should not be diagnosed during pregnancy except when the most stringent criteria are applied — a high fasting blood sugar (over 120 mg. per 100 cc. but preferably over 130 mg.) or a high three-hour level (over 120 mg). Even then an occasional case may show a return to normal after the puerperium.

In regard to the question of glycosuria during pregnancy, it is of interest that of the 4 patients who showed large amounts of sugar in the urine during the glucose-tolerance test, all but 1 had high blood sugars — over 180 mg. — at the time of the glycosuria. Figure 2 illustrates this fact. Glycosuria during pregnancy is usually ascribed to lowering of the renal threshold. From this figure it is readily apparent that significant glycosuria occurred at the time of or soon after a marked elevation in the blood sugar. Since the curve dropped to normal, it is justifiable to classify these curves as those of high alimentary hyperglycemia.

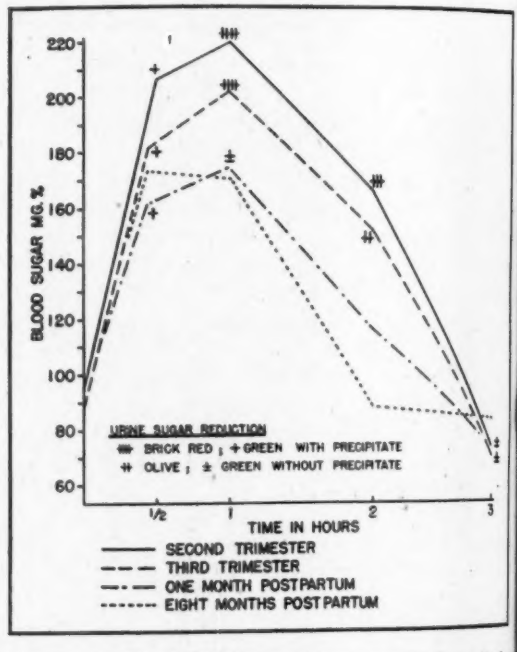

FIGURE 2. *Glucose-Tolerance Curves on the Same Patient at Various Times during Pregnancy and after Delivery. Note the association of marked glycosuria with hyperglycemia.*

SUMMARY AND CONCLUSIONS

Serial glucose-tolerance tests were done on 25 normal pregnant patients — at each trimester, immediately after delivery and several months post partum.

The fasting blood-sugar values were well within

normal limits. The peaks, too, were normal except in rare cases. The two-hour levels during pregnancy were abnormally high in a large percentage of cases (81 per cent). For this reason, a high two-hour level cannot be considered diagnostic of diabetes during pregnancy. On the other hand, the three-hour level was normal during pregnancy with one exception and may thus be more properly used as a criterion. The incidence of high two-hour values persisted immediately after delivery but dropped sharply after the puerperium. These findings indicate an adverse effect of pregnancy on carbohydrate metabolism.

Pregnancy glycosuria is probably due to a high alimentary hyperglycemia rather than to a lowering of the renal threshold.

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MECKEL'S DIVERTICULUM*

With Report of a Case of Intussusception Due to Its Invagination

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THE presence of an appendage or diverticulum in the small intestine was observed by Lavater[1] as early as 1672. In 1700, Littrê described a structure that was probably a true congenital diverticulum, but stated that the appendage lacked the usual muscular coats.[2] Both Ruysh and Mery recorded the phenomenon in 1701.[3] Ruysh was the first to name it "diverticulum," and his *Thesaurus Anatomicus* contains an illustration of this blind sac. Morgagni[4] in 1769 mentioned several cases of diverticulum of the ileum and suggested their embryologic origin, having previously noticed this type of diverticulum in geese. Sandifort[5] described a case of acute intestinal obstruction probably due to Meckel's diverticulum as early as 1793. But inasmuch as Meckel[6] was the first to give an extensive description of this structure, with an explanation of its origin and clinical importance, his name was given to this particular diverticulum of the ileum. He described it as an embryonic rest or remnant of the vitelline duct that had failed to become obliterated.

The earliest known case of an anatomic specimen of this appendage is that placed in the anatomical museum of St. Bartholomew's Hospital, London, in 1846.[7] The ornithologist Elliot Coues mentions finding this abnormality in birds and explains the variability of the site along developmental lines.[8]

In 1933, Harkins[7] published a report of 160 cases collected from the literature and added 2 of his own. A number of authors[9–19] have reported cases since this date, but little additional information is to be obtained from these cases beyond Harkins's conclusions. After reviewing the literature, he stated that Meckel's diverticulum occurs in normal people in 1.3 per cent of cases and is three times as frequent in males as in females. Many authors claim that 75 per cent are found in males. Meckel's diverticulum is found in 2 to 3 per cent of all autopsies. Intestinal obstruction from this abnormality occurs in an extremely small percentage of cases — 1.5 per cent according to Harkins, 3 per cent according to Richardson and 6 per cent according to Halsted and Lichtenstern.[2]

The obstruction may be brought about in any one of a number of ways. The obliterated omphalomesenteric duct in the form of a fibrous band may constrict the bowel. The tip of the diverticulum may attach itself to some point in the peritoneal cavity and act as a constricting band. A long, mobile diverticulum may tie a knot around a loop of free bowel, as in Dorling's[20] case, in which the ileum was thus involved. A long diverticulum — the average diverticulum according to Lamb[21] is 5 cm. long — may become adherent to the intestine or umbilicus and act as a band, causing strangulation or volvulus. The diverticulum may invaginate itself into the lumen of the bowel and start an intussusception, in which case a polyp is often present in the diverticulum. The first case of this sort was cited by Moroni[22] in 1898. A starting point may be the expulsion of a fecal mass or concretion from the lumen of the diverticulum into the intestine. The diverticulum becomes inverted and acts as a polyp, initiating an intussusception. The mesentery is usually dragged into the intussusception and the traction causes pain. McCann[2] states that to invert a Meckel's diverticulum must hang free. As a rule, there are secondary attachments to the intestine, mesentery or umbilicus. Peristalsis of the diverticulum itself may cause inversion with negative pressure exerted by the rapid flow of intestinal contents. Inflammation may cause swelling at the base of the diaphragm and inversion into the lumen of the intestine. The bowel attempts to expel it, and this causes bleeding and inversion of the mucosa and serosa.

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Meckel's diverticulum is usually found 30 to 45 cm. proximal to the ileocecal valve, but it may occur at any point between the pylorus and the ileocecal valve — as a rule in the lower fourth of the ileum. Ordinarily it arises from the bowel opposite the attachment of the mesentery, but it may arise at either side or between the two leaves. In infants, it is often found nearer the ileocecal valve, because the site

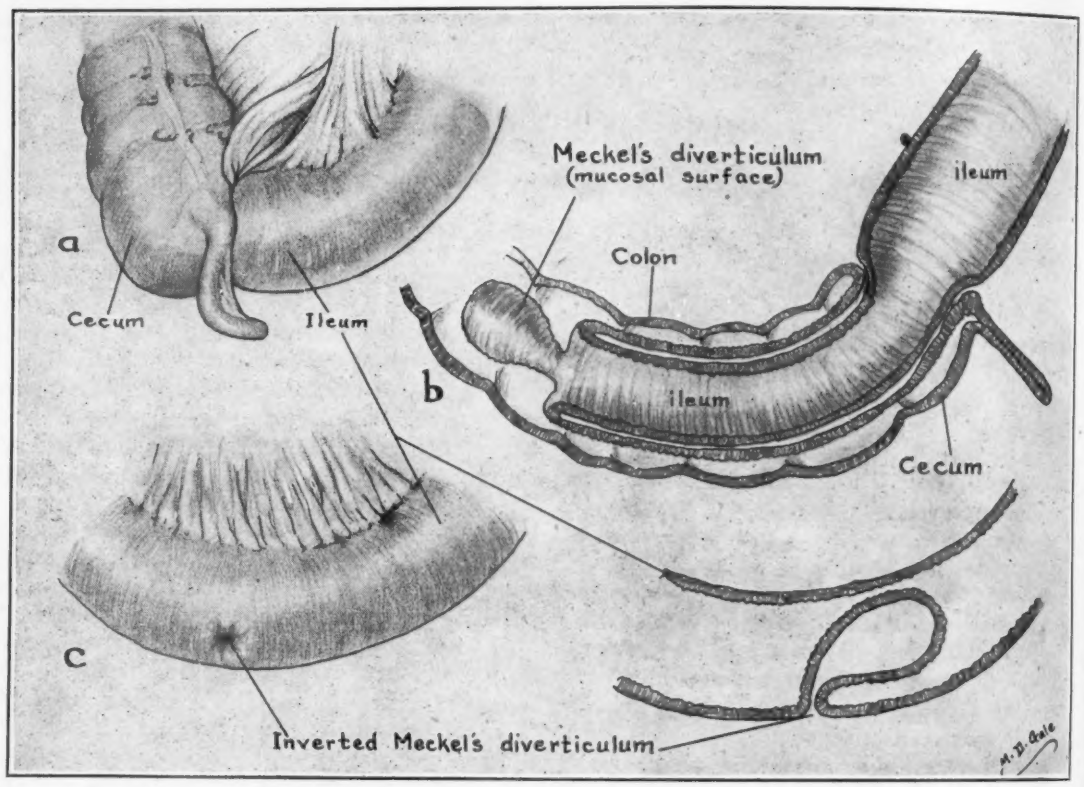

FIGURE 1.

depends on the amount of growth in the portion of the intestine proximal to the insertion of the omphalovitelline duct.

Thirty-seven cases of Meckel's diverticulum have been recorded at the Truesdale Hospital in 69,000 admissions. Thirteen of these were found at autopsy. Intussusception as a result of inversion of the diverticulum occurred in only 1 case, which is reported below.

CASE REPORT

A. S. (65619), a 7-year-old boy, was admitted to the Truesdale Hospital on July 10, 1944. The mother stated that symptoms began on the morning of July 8, when the patient complained of severe abdominal pain, accompanied by nausea and vomiting. He had been eating green apples, and the mother thought that he had an ordinary stomachache. The pain continued during the following day, along with the nausea and vomiting. Early on the morning of July 10, the family physician was called. By that time the patient was lethargic and cyanotic. There was an odor of acidosis on the breath, and the abdomen was distended. He immediately referred the patient to the hospital. There was no history of any previous similar attack.

Physical examination on admission showed a well developed, fairly well nourished, slender boy, complaining of severe abdominal pain. The lips were cyanotic, and the general condition was extremely poor. The temperature was 100.6°F., the pulse 130, and the respirations 22. The respirations were labored and slightly increased. There was a marked odor of acetone on the breath. The lungs were clear and resonant, and no rales were heard. The heart sounds were of fairly good quality, with a rapid rate. The abdomen was markedly distended and tympanitic to percussion, with generalized tenderness. No peristalsis was heard. The patient was thought to have acute intestinal obstruction from an unknown cause.

Urinalysis showed the slightest possible trace of albumin, large numbers of hyaline casts, a rare pus cell and mucus. The hemoglobin was 80 per cent, the red-cell count 4,440,000, and the white-cell count 14,650, with 69 per cent neutrophils, 20 per cent lymphocytes, 2 per cent large mononuclear leukocytes, 8 per cent band forms, and 1 per cent metamyelocytes.

The patient was extremely restless from the time of admission. The pulse was rapid and weak. He vomited at frequent intervals. A rectal tube was inserted, and 1000 cc. of 5 per cent glucose in saline solution was given by clysis.

At 2.20 p.m. the patient was taken to the operating room, where exploratory laparotomy was done under nitrous oxide, oxygen and ether anesthesia. The abdomen was opened through a right-rectus muscle-splitting incision. A considerable amount of foul-smelling fluid was present in the abdominal cavity. The small intestine was markedly distended and ballooned out of the abdomen. At that point, the patient vomited large quantities of foul-smelling fluid; suction was used, with a reasonably good result. His condition, however, was only fair.

By following along the small intestine in the direction of the cecum, a point of obstruction was located in the terminal ileum that had prolapsed through the ileocecal valve (Fig. 1*a*). The mechanism of the inversion and intussusception is shown in Figure 1*b*. The intussusception was reduced by expulsion and milking. At its apex was an inverted Meckel's

diverticulum. On reduction of the intussusception, a dimple was found in the ileum at the point where the diverticulum inverted into the lumen of the bowel (Fig. 1*c*). It was believed that a piece of green apple was the exciting cause of the intussusception. No polyp was present.

With considerable difficulty, the bowel was replaced in the abdominal cavity. During this procedure the patient's condition caused anxiety. Fluid was withdrawn from the stomach through a Levin tube. The pulse was of poor quality and the skin color fair. The patient's condition improved with the administration of oxygen and intravenous glucose. No further operative measures were attempted. The abdomen was closed with several mass interrupted nylon sutures through the peritoneum, muscle, fascia and skin.

At the close of the operation, which lasted for 25 minutes, the patient's condition was critical. In spite of oxygen and supportive treatment, it became steadily worse, and death occurred 1 hour after operation. Permission for an autopsy could not be obtained.

In these cases a definite history of previous abdominal crises is fairly frequent. In many there is a less severe attack a day or two before the final attack that sends the patient to the hospital. One explanation is that the diverticulum becomes inverted before the bowel becomes invaginated.

Colicky pain is present in almost every case reported. Vomiting is present in 92 per cent of cases, and is a cardinal symptom out of proportion to other symptoms. Tenderness and rigidity may not be present, even when the bowel is gangrenous. A mass is almost always present, usually in the right lower quadrant of the abdomen. The mass is rarely found palpable by rectum. There is little fever or elevation of the white-cell count. Melena may or may not be present. The condition is usually taken for acute appendicitis or for intussusception from other causes. Indications for operation are the same in both conditions, so that the differential diagnosis is of but academic interest.

Harkins[7] states that the mortality in children is 56 per cent — 36 per cent in those over five years of age and 74 per cent in those under one year of age. In many cases the diagnosis is made too late and operation is done only as a last resort when the patient is in extremely poor condition. The benefit of early operation is seen in the statistics from his analysis of 160 cases (Table 1).

Table 1. *Mortality According to Time of Operation.*

Period between Onset and Operation	No. of Cases	Mortality
hr.		%
Less than 12	9	22
12-23	12	42
24-47	26	31
48-72	15	47
Over 72	24	58

The treatment in the 24 operative cases at the Truesdale Hospital is shown in Table 2. In the case in which only appendectomy was done, the Meckel's diverticulum was noted, but it had a large base and was not excised. The case in which cholecystectomy was done was complicated by a three-month pregnancy.

Srigley,[11] who reports a series of 4 cases with recovery, states that if the patient is in poor condition and the diverticulum is not gangrenous, it should not be excised. The intussusception may be treated by simple disinvagination. Often, however, a one-stage or two-stage resection is necessary. Bowel resection is less dangerous than it is in cases of ordinary intussusception, because most of the patients are older. In Harkins's[7] series the average age was twenty-one years. Bickham[23] advises excision of the diverticulum by a transverse elliptical incision, which should not be deep enough to result in encroachment on the lumen of the bowel. The appendix should not be removed unless it is gangrenous or has thrombosed vessels. When resection of the Meckel's diverticulum is considered feasible, a stricture may result from inversion with a purse-string suture. After the diverticulum has been excised by a wedge-shaped incision, the intestine should be closed transversely to the long axis of the bowel. Harris[10] makes an appeal to the surgeon to direct his mind from appendicitis in cases that are not clear cut. In cases of this sort, if a simple appendectomy is performed, the patient is frequently again taken ill with an attack like the first one. Furthermore, pain caused by a diverticulum is often considered to be due to adhesions, enteritis or colitis or the pelvic organs are blamed.

Table 2. *Type of Operative Treatment.*

Operation	No. of Cases
Excision of Meckel's diverticulum only*	4
Excision of Meckel's diverticulum and resection of portion of ileum	1
Excision of Meckel's diverticulum and appendectomy	15
Appendectomy only	1
Cholecystectomy only	1
Repair of inguinal hernia	1
Reduction of intussusception only	1
	24

*In 1 case appendectomy had been done previously.

Summary

The literature on the subject of intussusception due to an invaginated Meckel's diverticulum has been reviewed, and an additional case is reported. On admission the cause of the obstruction presented a problem in diagnosis, which was revealed at laparotomy.

The mechanism of the inversion of the Meckel's diverticulum to produce intussusception is illustrated.

This case, which ended fatally, illustrates the need of early diagnosis and surgery, while the patient is still a good operative risk and also the need in any laparotomy of a search for a Meckel's diverticulum, especially in children.

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MEDICAL PROGRESS

SYPHILIS

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THE use of penicillin in syphilotherapy has become so widespread and the volume of literature regarding this drug so voluminous that this year's progress report on syphilis is being presented in slightly different fashion. A separate main division is devoted to penicillin and placed at the end of the report.

Public Health

Little literature has appeared during the past year regarding the public-health aspects of syphilis. This may be due to some degree of optimism resulting from the remarkable reduction in the incidence of venereal disease during World War II. Never in history has any nation been able to accomplish such a feat. Despite this fact, control of venereal disease in the civilian population continues to be a tremendous problem. Editorial comment[1] cautions against overoptimism regarding its eradication. The recent advances in therapy, although highly effective, will not eliminate venereal disease unless more vigorous epidemiologic control is maintained. The establishment of rapid-treatment centers in many parts of the country, with the assistance of the United States Public Health Service through funds made available by Congress, is an example of what should now be done on a broader scale. By present methods of administration, penicillin treatment requires a week or more of hospitalization, and sufficient beds are not available. Syphilis must be dealt with in the same fashion as any other highly communicable disease, with the co-operation of federal, state and local public health services, before the disease can be wiped out.

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Incidence of Syphilis

Statistics are available on the incidence of syphilis in the first six months of the fiscal year 1944–1945, from the Health Officers' Monthly Statements.[2] The total amount of syphilis (171,535 cases) reported in the United States was approximately 25 per cent less than that reported during the first half of the preceding fiscal year. During the same period of time, primary and secondary syphilis (36,445 cases) continued to appear at nearly the same rate of incidence as in the year before. The latter figure indicates that public-health control measures have been holding the incidence of new infections about stationary. The decrease in total syphilis reported suggests that the reservoir of old infections is gradually being brought under treatment. If this is true, there should be a decrease in the amount of late crippling disease encountered hereafter.

The results of the first statutory requirement for mass civilian blood testing are now available. In July, 1943, the Legislature of Alabama passed a law requiring all civilians between the ages of fourteen and fifty to have their blood examined for syphilis. In the first three counties surveyed,[3] it was found that 80 to 90 per cent of the population voluntarily appeared to be tested, indicating that law-enforcement agencies will have to play but little part in the program. The Whites showed 2.2 per cent positive tests, and the Negroes 20.5 per cent. This is the commencement of an ideal campaign to eradicate syphilis. It will be time-consuming and expensive, but if continued on a nation-wide basis

may well constitute one of the greatest single public-health measures in history.

Control Measures

A summary of the contribution of the military forces toward postwar control of venereal disease[4] indicates a number of advantages that should greatly benefit the civilian population. A tremendous number of physicians and lay personnel trained by and educated in the military services regarding the principles of venereal-disease control will return to civilian life and will be available as workers in connection with venereal-disease control programs. Several million men who have received intensive venereal-disease education will return and should raise the level of information among the general public. Mass wartime experiences with the remarkable advances in treatment have contributed greatly to the therapeutic armamentarium. These factors present an unusual opportunity to reduce the incidence of venereal infection to a more readily controllable minimum.

The growth of programs for the control of venereal disease in industry is an encouraging circumstance. Anderson[5] has surveyed the accomplishments in this field, pointing out the co-operative achievements by both labor and management, and outlines the basic principles set down for the management of syphilis in industry by the United States Public Health Service. Industrial physicians, by following the medical objectives of an industrial venereal-disease control program and co-operating with state and local health departments, can be of great service in establishing intelligent control measures.

An analysis of case-finding measures[6] in venereal-disease control indicates that in the area surveyed more persons with infectious lesions voluntarily applied for aid than were brought under care by any other means. This illustrates the value of community education and of publicizing the availability of services. Although mass blood testing on a large enough scale will uncover more syphilis than will any other method of case finding, it will be severely handicapped unless intensive community education is successfully carried out.

An interesting report[7] on the value of educational procedures among the general public has appeared. A county in Georgia with a population of approximately 40,000, half being Negroes, was selected. Adequate facilities for diagnosis and treatment were provided. The community was then made aware of the prevalence of venereal disease, the facilities available and the newer methods of therapy. Every measure was taken to encourage patients to seek examination, either by private physicians or at public clinics. It was found that of the patients who came voluntarily to public-treatment centers, 76 per cent were directed to the clinic by posters seen in public lavatories. Negro churches were utilized to disseminate information on venereal disease. An effort was made to encourage teaching on venereal disease in the schools for Negroes. Both these measures also proved of value, especially from the standpoint of improvement in knowledge of venereal disease. This report is an example of what can be accomplished even among the least privileged citizens.

Cost of Syphilis

Estimating with any accuracy the financial burden imposed yearly on the Nation by this single disease is probably a near impossibility. A study is available, however, on the economic cost of paresis.[8] The data were obtained from a survey of admissions to state, county, city, private and Veterans Administration hospitals. The annual direct cost to the taxpayer of maintaining paretics is over $11,000,000. The loss of income to the paretic patients themselves is estimated at $112,000,000 yearly for men alone. These figures are astounding, especially when one considers that paresis is only one of the late manifestations of syphilis and not the only cause of insanity due to the disease.

Experimental Studies

Aside from the vast amount of research related to penicillin in the treatment of syphilis, the main field of study has been related to hepatic dysfunction during the chemotherapy of syphilis. One investigator in particular, Dr. A. C. Ivy, has for many years made an intensive study of the physiology and diseases of the liver. The excretion of neoarsphenamine arsenic and Mapharsen arsenic in the bile of dogs with permanent bile fistulas has recently been investigated by Ivy and his co-workers.[9] It was found that 40 per cent of the arsenic in Mapharsen was recovered in the bile in forty-eight hours, whereas the same percentage from neoarsphenamine was recovered in seventy-two hours. The production of choleresis in dogs did not augment the rate of excretion of arsenic in bile, and arsenic excreted in the bile was not thoroughly absorbed from the intestine. The administration of sodium dehydrocholate tended to decrease the elimination of arsenic, and the administration of sodium glycocholate and taurocholate, which cause less choleresis, increased the elimination of arsenic in the bile. If the administration of dehydrocholic acid diminishes the hepatotoxic action of neoarsphenamine and Mapharsen, it apparently does not do so by increasing their excretion in the bile. It was thus concluded that these results obviously do not provide a clear and substantial rationale for the administration of bile acids with arsenicals. Further work by the same group of investigators[10] indicated that neoarsphenamine does not constantly depress the output of cholic acid in dogs with a permanent biliary fistula. Mapharsen did constantly depress this output in

such dogs when the animals were deprived of bile acids. The oral administration of dehydrocholic acid may assist in counteracting the hepatotoxic action of Mapharsen. These findings led to the suggestion that patients scheduled to receive intensive arsenotherapy should for two or three days prior to the initiation of therapy have a liver-function test and be given a diet known to affect favorably the liver's resistance to poisons. Adequate protein is the essential feature of this diet.[11] Other workers,[12] studying liver damage from Mapharsen in dogs, present evidence that the oral administration of methionine affords protection against hepatic injury following large doses of Mapharsen given to protein-depleted dogs. Clinical application of this work indicates that the oral administration of methionine and cystine are effective in delaying the onset of liver damage and in diminishing the severity of the hepatitis in human beings treated with neoarsphenamine, but has no effect on the total incidence of liver damage.[13]

It is obvious from the reports of these several groups of investigators that the hepatotoxic effect of organic arsenical compounds is not too well understood. Present indications are that less and less arsenic will be used hereafter, if penicillin proves to be all that is hoped. It seems unlikely, however, that a form of chemotherapy that has stood so well the test of time can be completely discarded, and it is to be hoped that further study of these drugs will be continued.

Serologic Problems

Considerable attention has been devoted during the past year to the blood serologic aspects of syphilis. There is a continuous striving to improve technic in the performance of blood tests and to eliminate the false-positive findings that are not infrequently encountered. But few of the reports in this field will be quoted, since much of the literature is of no direct value to the practicing physician. An excellent general discussion of the serologic findings in early syphilis is available.[14] It is pointed out therein that a limited serologic routine furnishes sufficient data for the usual diagnostic purposes and for the evaluation of clinical response to an established treatment. This justifies the performance of only one or two reliable tests, as carried out by most hospitals, state health departments and city laboratories.

The more highly refined quantitative tests are mainly valuable in gauging serologic response, such as a decreasing titer or the development of a serologic relapse. Their usefulness is thus limited to certain special situations and research projects. An extensive battery of different tests falls in the same category. Kahn[15] has published a short bulletin intended to serve as a practical guide to the technics of the various serologic tests that he has devised for the examination of serum and of spinal fluid. The bulletin also outlines the method of interpreting and reporting these tests. This is a useful manual, especially for those engaged in laboratory work.

The indications for quantitative serologic tests for syphilis were reviewed in this journal some months ago.[16] Briefly enumerated, these include the diagnosis of primary syphilis in dark-field-negative lesions, comparative reagin titers in mother and child when congenital syphilis is suspected and the detection of impending relapse, including the clarification of certain technical points. Quantitative procedures estimate the potency (amount of reagin), in the same general fashion as in the Widal reaction, by quantitative titration of the serums. Some authors[17] advocate general adoption of these procedures for use in the management of every phase of syphilis. Limitations of their usefulness, possible difficulties of interpretation in general practice and the expense involved seem to preclude their widespread application at the present time.

Still further refinements in serologic technics are the verification tests that have been devised to assist in identifying false-positive serologic reactions. After extensive experience, Rein and Callender[18] believe that the average serologist is not able to distinguish consistently between true-positive and false-positive reactions by the use of any verifying test yet devised. They consider these tests, for the present, in the experimental stage and stress the oft-repeated caution that a diagnosis of syphilis should not depend on serologic reactions alone. The ensemble of available data, before diagnosis, should include the history and physical examination, radiologic studies, spinal-fluid findings and examination of contacts and family, in addition to repeated serologic reports from more than one laboratory. Additional measures may be necessary to rule out nonsyphilitic diseases that may cause false-positive serologic reactions. A discussion of the principles of verification tests is available,[19] with a description of the chemical and immunologic aspects of the reactions of syphilitic and false-positive serums. The possibility of the application of verification tests to the development of a practical method of differentiation is encouraging.

False-Positive Reactions

General considerations on the occurrence of false-positive reactions for syphilis have been reviewed by Rein and Elsberg.[20] Most of this report is concerned with the so-called "biologic" false-positive reactions, occurring in such disease as vaccinia, upper respiratory infections, filariasis, Weil's disease, malaria, typhus fever and leprosy. The incidences of false-positive reactions in these diseases varied from 11.3 per cent in filariasis to 85.0 per cent in leprosy. Davis[21] reports a review of the literature regarding false-positive reactions for the years 1930-1943. He outlines the long list of diseases in which

such reactions have been reported. Such diseases have been listed in these columns several times in recent years and will not be repeated at this time. It seems altogether possible that some of the diseases that Davis includes may not have been responsible for the false-positive reactions, since their incidences are so extremely low. Serologic tests for syphilis are subject to marked variations in sensitivity, which may account for a large proportion of discrepancies in published reports of false-positive reactions in various diseases. Technical errors also must not be forgotten. Davis stresses the importance of a sufficient period of observation, except during pregnancy, before making a final diagnosis in questionable cases. He indicates a period of three months, but a much longer time is often necessary. A point that is stressed is that a positive serologic test for syphilis is not an emergency. This is obviously true in the absence of infectious lesions, but neither should positive tests be lightly regarded, as so often occurs. Even the best trained men in other fields of work, from time to time, pay too little attention to positive serologic tests for syphilis and do not seek expert advice until the development of clinical lesions. These may be of minor clinical import, but they are not infrequently most distressing to the patient and are completely unnecessary.

Numerous reports on the effect of malaria on serologic tests for syphilis continue to appear. In a series of more than eight thousand tests carried out by six standard technics, the Hinton flocculation test for syphilis showed the smallest proportion of false-positive reactions in malaria of any of the serologic technics employed.[22] It was stated that in the absence of continued evidence of malarial infection, the persistence of positive reactions to any serologic test beyond a period of six weeks should arouse the suspicion of syphilis. This is a shorter time period than some observers have reported in the past, but agreement with a six-week time factor is available.[22, 24] Spinal-fluid examinations made on 100 malaria patients within forty-eight hours after hospitalization were found to be essentially normal.[24] Slight deviations from normal occurred in the spinal-fluid findings of 4 cases, but these were not indicative of syphilis and there were no neurologic symptoms.

Favorite[25] has continued his study of false-positive reactions for syphilis due to smallpox vaccination (vaccinia). Five hundred and twenty-five cases are included in his latest report. Of 202 patients on whom a single blood examination was performed, 11.8 per cent gave positive reactions. These appeared within two weeks of the date of vaccination and usually disappeared within two months, but a few weakly positive reactions persisted for as long as four months. A second group of 323 persons, who had had smallpox vaccinations several months previously, were given typhoid vaccine and tetanus toxoid to determine the role played by immunization. No significant bearing on the production of false-positive serologic reactions for syphilis was observed. Recently vaccinated persons who had exhibited a temporary false-positive reaction did not have a reactivation of the reaction following typhoid and tetanus immunizations. It was found, however, that if these patients were revaccinated for smallpox within seven months, they might again present a weak serologic false-positive response for syphilis, even though the second vaccinia reaction was of the immune type. In a series of 129 patients with vaccinia reported during the past year, 44.9 per cent showed falsely doubtful or positive tests with one or more serodiagnostic procedures.[26] This is an extremely high incidence compared with that in most reports in the literature. The application of the Kahn verification test to post-vaccination false-positive serologic reports has apparently been useful.[27]

In contrast to Favorite's[25] experience with tetanus toxoid, other observers[28] report 8 cases in which biologic false-positive serologic reactions were considered to be directly related to stimulating doses of tetanus toxoid. Infectious hepatitis was apparently responsible for fifteen false-positive or doubtful serologic reactions after the clinical appearance of jaundice in 63 patients among allied military personnel in Sicily.[29] All but one of the reactions were negative by the seventh week. During an epidemic of upper respiratory infections among troops in this country, 100 patients were found to have positive serologic reactions for syphilis.[30] All had received routine Army immunizations from five days to four months previously. In 90 per cent of these cases, the false-positive nature of the serologic reactions was established. That the upper respiratory infection played any appreciable role in these false-positive cases seems doubtful.

Clinical Problems

The third edition of *Modern Clinical Syphilology*, by Stokes,[31] appeared some months ago. This superb book on the diagnosis and treatment of syphilis has been brought up to date by the rewriting of a large proportion of the text and the addition of chapters on penicillin, military medicine and public health. Stokes's favorite and effective "thumbnail summaries" are featured as before. This magnificent work should be in the possession of everyone who is interested in syphilis.

Reinfection

On numerous occasions in the past, the criteria for the cure of syphilis and for the diagnosis of reinfection with the disease have been discussed in these columns. The advent of intensive treatment programs and more lately the startling effectiveness of penicillin have necessarily brought about changes in the concept of such reinfection. It becomes increasingly difficult to differentiate relapse and re-

infection. The possibility of superinfection although less likely, should always be kept in mind. Cases of supposed reinfection are five to ten times as frequent as in previous years.[32] The difficulty of adequately differentiating reinfection and relapse is illustrated by the variety of criteria that can be found postulated in the literature. The simplest criteria merely require proof of syphilis prior to the occurrence of the supposed second infection. This proof must consist of demonstration of spirochetes in the lesion or the occurrence of a positive blood serologic test and should not depend on clinical judgment alone. After an interval following antisyphilitic treatment, and at a site other than that of the primary lesion of the first infection, there must develop a new lesion with the characteristics of a chancre and in which spirochetes can be demonstrated. From these simplest criteria, one can go to the opposite extreme of the seventeen complex criteria that are proposed by Stokes. In some diseases, reinfection can be considered a proof of cure of a preceding infection. Moore[32] points out that this is not tenable with syphilis in human beings. After discussing the criteria of reinfection at some length, he states this opinion that, at the present time, re-infection cannot be differentiated from relapse on any grounds on which two or more observers would agree. He believes that this differentiation must rest on clinical impressions. This does not constitute proof. Moore points out that until order is brought out of the present confusion, all supposed reinfections must be regarded as treatment failures. This is especially applicable to the evaluation of new drugs and new treatment programs.

Syphilis of the Mouth

The various forms of syphilis of the mouth are discussed in some detail by Wilson, Dunning and Fox.[33] It is pointed out that the dentist has a large responsibility in discovering oral manifestations of systemic diseases. The oral lesions of primary, secondary and late syphilis, both acquired and congenital, are described, including those of the lips, tongue, oral cavity, teeth and palate. A differentiation from the usual nonsyphilitic diseases that may simulate syphilis of the mouth is given. Included in this list are aphthous stomatitis, herpes simplex, herpes zoster, pemphigus, erythema multiforme, lichen planus, lupus erythematosus, leukoplakia, lupus vulgaris, Vincent's infection, granuloma pyogenicum, epithelioma, lingua plicata, lingua geographica, dysvitaminosis, hypovitaminosis, avitaminosis, pellagra, fungous infections and perlèche. A word of caution has been written against overlooking the possibility of cancer of the tongue in patients with syphilis.[34] It is pointed out that cancer can exist in conjunction with syphilis of the tongue in any stage, but may be overshadowed by syphilis when the cancer is early. More frequent biopsies of possibly suspicious areas would provide an earlier diagnosis of cancer and a greater percentage of cures. Needless to say, adequate treatment for coexisting syphilis must be diligently pursued.

Visceral Syphilis

O'Leary and Ockuly[35] point out that the only satisfactory method for determining that a lung lesion is syphilitic is a therapeutic test. The symptoms, physical signs and radiologic appearance of pulmonary syphilis are nondistinctive. When, however, a patient with positive serologic reactions is dyspneic, coughs, raises bloody sputum, has chest pain, is weak and loses weight and when physical and radiologic examinations indicate a nodular or infiltrative pulmonary process, one may presume that the lung lesion is syphilitic. If these signs and symptoms disappear after a course of therapy and do not recur, the opinion that the condition was syphilitic is tenable. In a series of 60 patients at the Mayo Clinic in which syphilis of the lung was considered a possibility, this diagnosis was substantiated in only 4 cases. Syphilis of the lung rarely recurs following involution under treatment.

Among 1705 consecutive adults admitted to a tuberculosis sanatorium for Negroes, 29.7 per cent were found to have positive serologic reactions for syphilis.[36] These patients were studied from a number of viewpoints without significant findings regarding a biologic relation between the two diseases. It has long been believed that the prognosis in such circumstances depends entirely on the tuberculosis, an opinion that was substantiated by this investigation.

Syphilis in diabetes has been studied by Perkin[37] over a five-year period. Among 550 diabetic patients comprising the series, 54 possible cases of syphilis (9.8 per cent) were found. The diabetes in the syphilitic group tended to be unusually mild. It is the author's opinion that syphilis, as well as all other so-called "causes" of diabetes, is merely a precipitating factor in the potential diabetic patient. These cases were observed for varying periods up to twelve years for changes in diabetic status. It is stated that there is some relation between latent syphilis in the diabetic patient and the occurrence of gangrene, but the supporting evidence is not too conclusive.

Neurosyphilis

An interesting study concerns the incidence of asymptomatic neurosyphilis in young men.[38] The data were obtained from the spinal-fluid findings of 3000 syphilitic military selectees passing through an induction station. Sufficient pathologic changes to cause rejection were found in 294 (9.5 per cent), all but 15 of whom had positive Wassermann reactions in the spinal fluid. The second most frequent abnormality was an increased globulin (174 cases). The colloidal gold curve was elevated in 92 cases,

but only 38 of the 3000 spinal fluids showed an increase in cell count.

A strikingly low percentage of lumbar-puncture headaches were observed in an army camp.[39] During a five-month period, lumbar punctures were performed on 2217 syphilitic patients, among whom only 15 (less than 1 per cent) developed puncture headache severe enough to require bed rest. The fluids were collected rapidly; the patients immediately got on their feet and were instructed to keep active and not to lie down. It was believed that more nearly normal intracranial pressure was maintained by the erect position, thus preventing oversecretion and compensatory hypertension, with resultant headache.

An excellent review and clinical survey of symptomatic neurosyphilis has been published by Solomon, Moore, O'Leary, Stokes and Thomas.[40] It is pointed out that the symptoms of syphilis of the nervous system may simulate all forms and varieties of neuropsychiatric disorder. Obviously, then, in any disorder suggesting organic involvement of the nervous system neurosyphilis must be ruled out. These authors divide neurosyphilis into two main categories: meningovascular neurosyphilis, which produces little or no irreparable damage to nerve cells, except secondarily by interruption of the blood supply; and parenchymatous neurosyphilis. Trivalent arsenicals and bismuth are of great benefit in meningovascular disease but not in parenchymatous neurosyphilis. Obviously, the clinical distinction between the two is of great importance. The symptoms of neurosyphilis are directly dependent on the site of involvement as well as on the extent of the lesion. The clinical manifestations of the various types of neurosyphilis — syphilitic meningitis, meningovascular neurosyphilis, tabes dorsalis, general paresis, vascular neurosyphilis and congenital neurosyphilis — are described. Tabes dorsalis and its sequelae are discussed at some length. The great variety of symptoms that may be due to general paresis are emphasized. Attention is directed to the fact that early stages of dementia paralytica are too easily and too frequently confused with psychoneurosis. The treatment of each variety of neurosyphilis is discussed. It is well known that parenchymatous neurosyphilis may be accompanied by negative spinal-fluid findings. Seven cases of general paresis were recently reported in which a negative spinal-fluid Wassermann reaction[41] occurred. Several of these patients exhibited completely negative spinal fluids.

(To be concluded)

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MASSACHUSETTS MEDICAL SOCIETY

SPECIAL MEETING OF THE COUNCIL

January 9, 1946

A SPECIAL meeting of the Council was called to order by the president, Dr. Reginald Fitz, on Wednesday, January 9, 1946, at 3:00 p.m., in John Ware Hall, 8 Fenway, Boston. Dr. Michael A. Tighe served as secretary.

The following councilors were present:

BERKSHIRE
I. S. F. Dodd
P. J. Sullivan

BRISTOL NORTH
W. H. Allen
R. M. Chambers
W. J. Morse
W. M. Stobbs

BRISTOL SOUTH
J. C. Corrigan
E. D. Gardner
C. C. Tripp

ESSEX NORTH
E. S. Bagnall
R. V. Baketel
Elizabeth Councilman
F. W. Snow
C. F. Warren

ESSEX SOUTH
P. P. Johnson
A. E. Parkhurst
W. G. Phippen
H. D. Stebbins
P. E. Tivnan
C. F. Twomey
C. A. Worthen

FRANKLIN
H. M. Kemp

HAMPDEN
W. A. R. Chapin
E. C. Dubois
G. L. Gabler
Charles Jurist
A. G. Rice
J. A. Seaman

HAMPSHIRE
W. M. Dobson

MIDDLESEX EAST
R. W. Layton
M. J. Quinn
R. R. Stratton

MIDDLESEX NORTH
J. J. Cassidy
W. M. Collins
D. J. Ellison
A. R. Gardner
W. F. Ryan
M. A. Tighe

MIDDLESEX SOUTH
E. W. Barron
Harris Bass
G. F. H. Bowers
Alice M. Broadhurst
R. N. Brown
R. W. Buck
J. F. Casey
C. W. Clark
J. A. Daley
C. W. Finnerty
H. Q. Gallupe
V. A. Getting
H. G. Giddings
Eliot Hubbard, Jr.
F. R. Jouett
A. A. Levi
A. N. Makechnie
J. C. Merriam
Dudley Merrill
C. E. Mongan
G. M. Morrison
J. P. Nelligan
Dwight O'Hara
Fabyan Packard
Max Ritvo
E. H. Robbins
M. J. Schlesinger
H. P. Stevens
Hovhannes Zovickian

NORFOLK
B. E. Barton
Carl Bearse
J. H. Cauley
D. J. Collins
G. L. Doherty
H. M. Emmons
Susannah Friedman
H. B. Harris
P. J. Jakmauh
C. J. Kickham
C. J. E. Kickham
D. S. Luce
F. P. McCarthy
H. L. McCarthy
F. J. Moran
Hyman Morrison
D. J. Mullane
J. J. O'Connell
D. D. Scannell
L. A. Sieracki
Kathleyne S. Snow
S. L. Skvirsky
N. A. Welch

NORFOLK SOUTH
D. L. Belding
Harry Braverman
Frederick Hinchliffe
D. B. Reardon

PLYMOUTH
P. B. Kelly
P. H. Leavitt
B. H. Peirce
W. H. Pulsifer

SUFFOLK
W. H. Blanchard
W. J. Brickley
W. E. Browne
A. J. A. Campbell
David Cheever
N. W. Faxon
Reginald Fitz
Maurice Fremont-Smith
Channing Frothingham
F. C. Hall
John Homans
A. A. Hornor
R. I. Lee
C. C. Lund
W. J. Mixter
Donald Munro
H. F. Newton
R. N. Nye
F. R. Ober
F. W. O'Brien
J. P. O'Hare
L. E. Parkins
L. E. Phaneuf
Helen S. Pittman
H. F. Root
R. M. Smith
J. J. Todd
S. N. Vose
Conrad Wesselhoeft

WORCESTER
A. W. Atwood
George Ballantyne
F. P. Bousquet
E. J. Crane
J. J. Dumphy
W. J. Elliott
John Fallon
L. M. Felton
J. C. McCann
H. L. Paine
R. J. Ward
B. C. Wheeler

WORCESTER NORTH
H. C. Arey
C. B. Gay
G. P. Keaveny
J. V. McHugh

The business before this meeting was the report of the Postwar Planning Committee, which concerned itself with certain basic principles considered essential to a medical-care plan, the Wagner-Murray–Dingell Act (Senate 1606), and the Maternal and Child Welfare Act of 1945 (Senate 1318).

The report of the committee presented by Dr. Howard F. Root, Suffolk, chairman, as amended and as adopted by the Council, is as follows:

The committee and its subcommittees have held many meetings. The Subcommittee on Medical Economics has given intensive study to the Wagner Act (Senate 1606), to the Maternal and Child Welfare Act of 1945 (Senate 1318), to certain basic principles considered essential to a medical-care plan and to various methods of payment for medical services. For the purpose of informing the Society as a whole, its work has been summarized in the *New England Journal of Medicine*, issue of January 3, 1946.

The committee believes that the following basic principles are essential to a medical-care plan in a free society:

1. The objective of adequate medical care in our free society is to make available to everyone — regardless of race, color, creed, financial status, or place of residence — every known essential preventive, diagnostic and curative medical service of high quality. The attainment of such medical care must necessarily be an evolutionary process which will require the co-operation of all concerned over a period of years.

2. The success of any plan for medical care is dependent on the mutual co-operation of the public, those rendering professional services and the administrative agencies. This co-operation can be obtained only if those rendering the services are convinced that they will have a continuing authoritative voice in the formulation and execution of policies and plans, thereby assuming their proper share of responsibility.

3. Provision of adequate medical care for those unable to obtain it by voluntary prepayment plans or by direct payment is the responsibility of the local or state government. Part of the burden of this responsibility may be assumed by charitable agencies. Federal grants-in-aid to state programs administered by state boards of health is an acceptable method of helping to meet this responsibility.
4. The medical care of those who are able to purchase it by voluntary prepayment plans or by direct payment is the responsibility of the individual.
5. Eligibility for receiving benefits under a program aided by federal grants should be determined by the individual states.
6. The patient shall have free choice of his physician, group of physicians, clinic or hospital from among those participating in any plan, provided that the physician, group of physicians, clinic or hospital selected shall have the right to refuse or to accept the patient.
7. Physicians and other qualified persons rendering medical care shall receive adequate remuneration for their services.
8. The physician shall be free to elect or reject without prejudice participation in a medical-care plan. The rights of the physician as to the choice of methods by which he is to be paid shall be fully protected.

The committee recommends the adoption by the Council of the above basic principles as essential to the development of any successful medical-care plan and, as guides by which to evaluate medical-care plans that may be proposed in future, with the understanding that changing conditions may require their later revision.

Dr. Root moved the adoption of this recommendation. This motion was seconded, and it was so ordered by vote of the Council.

The Maternal and Child Welfare Act of 1945 (Senate 1318) has been carefully studied by the subcommittee. Although this subcommittee is in complete agreement with the objectives stated in the introduction to the bill, "To provide for the general welfare by enabling the several states to make adequate provision for health and welfare of mothers and children and for services to crippled children," it finds serious objections to the bill as written. Some of the more important of these are as follows:

(1) The bill makes no adequate provision for general public-health programs that are more fundamental than this specialized legislation.

(2) Services and facilities are available to all who elect to participate, regardless of economic status. (This violates Basic Principles 4 and 5.)

(3) The public deserves a reasonable estimate concerning the ultimate cost of this proposed legislation. Experience and such factual data as are available indicate an ultimate annual budget approximating one billion dollars. This should be clearly recognized in any consideration of the bill.

(4) Fee-for-service method of payment is restricted to consultation or emergency visits and is not ordinarily available to practitioners or specialists.

(5) The bill does not make clear just who is to decide the fee for a given service that would be considered adequate remuneration in an individual state, nor does it make provision for variable fees to meet the differing costs in the several states.

(6) The bill does not prohibit professional personnel, groups or institutions rendering service under the program from accepting supplemental payment from or on behalf of patients.

(7) The bill does not provide for payment to groups of physicians, clinics or hospitals providing professional services.

(8) The bill does not emphasize the desirability of full utilization and further development of existing services and facilities.

(9) The bill does not emphasize the necessity of restricting the development and expansion of a state program to the capacity of available administrative and professional resources.

(10) This bill does not provide the professions with a continuing authoritative voice in the formulation of policies and plans. Such committees as are selected under the provisions of the bill may represent the attitude of the administrator rather than that of a given profession or group. (This violates Basic Principle 2.)

(11) The bill does not provide the facilities whereby the opinions of both the federal and state advisory committees are made available to the public.

(12) Designation of the Children's Bureau as the administrative agency does not adequately assure proper integration of the health activities of the federal government.

For these reasons the committee believes that this proposed act does not represent the best form of legislation for the purposes for which it was written. The committee recommends the adoption of this report on the Maternal and Child Welfare Act of 1945 (Senate 1318).

Dr. Root moved the adoption of this recommendation. This motion was seconded, and it was so ordered by vote of the Council.

Senators Wagner and Murray introduced the National Health Act of 1945 (Senate 1606) in the Senate on November 19, 1945, the day of the reading of the President's message to Congress.

Title I of this bill authorizes grants-in-aid to states to (A) extend the public-health services, (B) increase maternal and child health services and (C) provide medical care for the needy.

Title II makes provision for full medical, dental, nursing and laboratory care and hospitalization for those able to pay for such services.

Because of the different provisions of the bill, it is desirable to discuss each part separately.

Title I. Grants to States for Health Services

Part A. *Grants to States for Public-Health Services*

The Massachusetts Medical Society cites the progressive leadership that the physicians of New England have always shown in the development of public-health enterprises, and its adoption as a principle the making available to everyone every known essential, preventive, diagnostic and curative medical service of high quality. We do approve in general this part of the bill as written, with the following exceptions:

(1) The responsibility for the training of personnel should be the duty of the states and not that of the federal government, as designated in Section 314*A*.

(2) Provision should be made which would ensure proper representation of professional organizations on the advisory council (Section 314*F*).

(3) Responsible private agencies, such as Blue Cross and Blue Shield, should be included among those with whom the state health agency may make working agreements (Section 314*H*).

(4) The state administrating agency should be required to consult with professional advisory committees before issuing regulations, which in turn should be issued only after due notice (Section 314*H*).

Part B. *Grants to States for Maternal and Child Health Services*

It is the feeling of this committee that the objectives of this part of the bill are more clearly and completely covered in Senate Bill 1318. Earlier in this report we have stated the reasons for our disapproval of that bill as written.

Part C. *Grants to States for Medical Care of Needy Persons*

We approve of federal grants to aid the several states in assuming their responsibility of providing medical care for those unable to pay for such services. We approve in general of this part of the bill as written with the following execptions:

(1) We believe that all federal grants-in-aid for the medical care of needy persons should be made by the United States Public Health Service to state departments of public health until such time as these grants may be made by a federal Department of Public Health.

(2) We disapprove of the provision that places the responsibility in the Social Security Board on the federal level and in the Department of Welfare on a state level (Section 131).

(3) This Society through its Blue Shield has approved of the service principle rather than the payment to patients of cash benefits for medical care. We disapprove of the provision in the bill of making cash payments to individuals for this purpose.

TITLE II. PREPAID PERSONAL HEALTH-SERVICE BENEFITS

This is compulsory health insurance. It would be very costly to carry out the provisions of this section of the bill, and there is nothing to intimate what this will add to the burden of the taxpayer or how the money will be raised. Since we believe that the payment for medical care of those able to pay for such services by direct payment or on a prepayment basis is the responsibility of the individual and that with the extension and development of voluntary prepayment or other plans particularly adapted to certain areas, medical care of the highest quality can be obtained at a reasonable cost, we disapprove of this section.

* * *

The Committee on Postwar Planning recommends that the Council direct the president of the Society to appoint a representative and an alternate to appear before the appropriate committees of the Congress of the United States for the purpose of making known the views of the Massachusetts Medical Society with regard to the Wagner-Murray-Dingell Bill (Senate 1606) and the Maternal and Child Welfare Act of 1945 (Senate 1318).

Dr. Root moved the adoption of this recommendation. This motion was seconded, and it was so ordered by vote of the Council.

The committee further recommends that the Council send copies of the article "Information on the Deliberations of the Subcommittee on Medical Economics," together with the action of the Council of the Massachusetts Medical Society, to all the members of the Congress of the United States, to the members of the Massachusetts Legislature and to the newspapers of the Commonwealth.

Dr. Root moved the adoption of this recommendation. This motion was seconded, and it was so ordered by vote of the Council.

It was moved by Dr. Leroy E. Parkins, Suffolk, and seconded, that the members of the various committees who had spent so many long hours of vigilant effort in preparing this report be given a vote of thanks. This motion was amended so as to include the President for his expert guidance. The motion was adopted by vote of the Council.

The Council adjourned at 6:35 p.m.

MICHAEL A. TIGHE, *Secretary*

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

BENJAMIN CASTLEMAN, M.D., *Associate Editor*

EDITH E. PARRIS, *Assistant Editor*

CASE 32101

PRESENTATION OF CASE

First admission. A sixty-five-year-old unemployed man entered the hospital because of a chronic draining ulcer of the lower lateral aspect of the right leg.

He had first been seen in the Orthopedic Clinic thirty-two years previously for a partially healed fracture of the right tibia just above the ankle. At that time an arthroplasty was performed and a metal plate was applied to the fibula. The ankle functioned well until about a year before admission, when a bruise developed over the right external malleolus after a minor injury. This gradually spread, and nine months later the skin broke down; there was constant drainage from the area thereafter. In the five years prior to admission he was seen many times in the Medical Clinic for a variety of complaints. Early in this period he had an episode of weakness, unproductive cough and fever lasting six weeks. His temperature during that time averaged 101°F. There was no sweating. He was well for several months and then had an attack of "bronchitis," during which he had chills, a fever rising to 104°F. and blood-streaked sputum for a period of three weeks. In addition to these febrile attacks he developed mild dyspnea on exertion and swelling of the ankles, most pronounced in the evening. At the same time he was troubled by difficulty in starting the urinary stream and by nocturia (one or two times). There were also numerous episodes of vague epigastric distress, with sensations of fullness and discomfort, sometimes accompanied by shortness of breath. X-ray examination of the chest at this hospital four years before admission showed clear lung fields. The cardiac pulsations were said to be extremely small. An electrocardiogram showed a PR interval of 0.16 second, a flat T_1, inverted T_2 and T_3 and a low T_4. The blood pressure on three occasions was 112 systolic, 90 diastolic, 100 systolic, 70 diastolic, and 80 systolic, 70 diastolic. The liver was palpable three fingerbreadths below the rib margin. He received 0.1 gm. of digitalis daily during the four-year period before admission, but continued to have episodes of ankle edema, swelling of the lower abdomen and dyspnea on moderate exertion. He stayed in bed for one to six weeks at times when these symptoms were at their worst.

Physical examination showed marked pigmentation and a 1-cm. ulcer on the lateral aspect of the right fibula. The head of a screw was visible in the floor of the ulcer. There were scattered rales at the lung bases. The heart was normal.

The temperature was 97°F., the pulse 80, and the respirations 20. The blood pressure was 120 systolic, 88 diastolic.

An x-ray film of the ankle showed bone absorption and periosteal proliferation about the metal plate. The lower end was free in the soft tissues. A chest plate showed hazy density of the left lower lung field. The left leaf of the diaphragm could not be visualized. The heart was in transverse position and appeared slightly enlarged to the left. There was no change in the electrocardiographic findings from the previous examination.

The bone plate was removed, and the wound healed slowly. A thoracentesis on the left side yielded 35 cc. of clear yellow fluid, which was negative for tumor cells; a guinea pig inoculated with it failed to develop tuberculosis.

Second admission (five months later). One month prior to this admission the patient developed pain in the left side of the chest, which persisted. It began at a point just below and lateral to the nipple and extended to a point just below the angle of the scapula. At first it was aggravated by deep respiration, but it was gradually reduced to a dull, constant ache. He lost 10 pounds in the month prior to entry. There was no cough or sensation of fever, although exertional dyspnea persisted.

On examination of the chest there were flatness and absent tactile fremitus and greatly diminished breath sounds over the lower half of the left chest. The temperature was 100°F.

Seven hundred and twenty cubic centimeters of bloody, cloudy fluid was removed through the ninth left intercostal space. The patient was discharged unimproved except that his temperature was normal on the fifth hospital day.

Third admission (six months later). The chest pain persisted after discharge except for periods of one to three days after each of four thoracenteses performed in the Emergency Ward. They yielded diminishing amounts of fluid, and after each a friction rub was heard. There had been no further weight loss, nor had cough or other symptoms developed.

The trachea was deviated slightly to the left. The first heart sound at the apex was slapping in character. The pulmonic second sound was split, giving an apparent systolic gallop. The left side of the thorax was flatter than the right and moved

poorly. The percussion note in the left axilla and lower half of the left chest was flat; breath sounds and tactile fremitus were diminished. The left side of the diaphragm did not appear to move. The liver was palpable two fingerbreadths below the ribs. X-ray examination revealed a markedly thickened pleura on the left side, with probable partial underlying collapse of the left lobe. The heart was displaced to the left, and there was a small amount of free fluid just above the diaphragm.

Biopsy of the left pleura showed slight chronic inflammation in dense collagenous tissue. Ten days after the operation he had a chill, with a rapid pulse and a temperature of 102°F., followed by a dry painful cough and pain on deep inspiration. There was slight ankle edema. No tenderness was elicited in the thighs or calves. The serum protein was 5.8 gm. per 100 cc., with an albumin of 2.4 gm. and a globulin of 3.4 gm. The prothrombin time was 28 seconds (normal, 22 seconds). The febrile episode lasted three days. He was discharged on the twenty-eighth hospital day.

Fourth admission (four months later). Soon after discharge the nocturia gradually increased so that he urinated every two hours. There were dribbling and some incontinence, followed by dysuria. The prostate was symmetrically enlarged to twice its normal size. A transurethral resection for benign prostatic hyperplasia was performed. Postoperatively he had an immediate episode of nervousness and shaking accompanied by cyanosis but without a chilly sensation, dyspnea or pain. The temperature rose to 104°F. This lasted only a few hours. He was discharged on the fifth postoperative day.

Final admission (two years later). He was apparently well until three days before re-entry, when he developed cough, with malaise and increasing fever.

On physical examination the lips were moderately cyanotic and the skin was flushed and hot. He was breathing hard at a rate of 40 respirations per minute. The right lung over the midportion posteriorly was dull to percussion, with bronchial breathing and inconstant rales in this area. The left cardiac border was 11 cm. to the left of the midsternal line. The rhythm was irregular, and the rate rapid.

The red-cell count was 5,450,000, and the hemoglobin 16.9 gm. The white-cell count was 16,400, with 92 per cent neutrophils, band forms predominating. The urine had a specific gravity of 1.018 and gave a ++ test for albumin. The sediment contained 25 white cells and a rare red cell per high-power field.

The temperature was 104°F., and the pulse 120. The blood pressure was 100 systolic, 60 diastolic.

X-ray films revealed an area of increased density in the right upper lung field, probably located in the dorsal division of the lower lobe. There was also an ill defined density in the left lower lung field, with apparent decrease in the size of the lef lower lobe and also a line of density extending alon the lateral chest wall.

The patient was started on penicillin (32,00 units every two hours). On the day after admissio the temperature had fallen to 100°F. and the respi rations to 25. The patient felt better but wa coughing up bloody sputum and had a pulse of 11 despite 0.8 mg. of Cedilanid intravenously and 0. mg. of Purodigin over a twelve-hour period. Ther was less dullness posteriorly but there were in creased fine rales throughout most of the righ chest. Abundant Type 3 pneumococci were isolate from the throat.

Two days later the temperature was back t 102°F. and he still had rales and was coughing u blood-streaked sputum. Sulfadiazine was starte in addition to penicillin. On the seventh hospita day he developed a generalized itching erythemato rash. There was considerable exfoliation over th arms. The pharynx was normal. Sulfadiazine wa stopped. Subsequently, the rash improved bu the patient's condition continued to deteriorate He remained cyanotic. He developed shifting dul ness in the abdomen, and the liver was palpabl two fingerbreadths below the ribs. Rales an rhonchi were constant in all lung fields.

On the eighteenth hospital day, just after he ha been taken off the bed pan, he gasped, fell back o the pillow deeply cyanotic and died.

Differential Diagnosis

Dr. Donald S. King: Four organs were involv in this long story: they took out a benign prosta and then there were three. The prostate explai satisfactorily the urinary symptoms. The remai ing involved areas are the leg — with the fractur many years before and the infection recently occu ring around the bone plate — and the lungs an the heart, both of which had given symptoms f four years, long before the recurrence of symptom in the leg. Of course, the temptation, when one presented with this story, is to try to combine a three systems in one diagnosis and say that th chest symptoms were embolic phenomena, with th source in the infected area in the leg. But as w shall see, such an explanation is not sufficient t cover all the findings.

So far as the pulmonary symptoms are concerne there were episodes of fever, and it was real feve sometimes with chills. At times the high tempera ture persisted for as long as six weeks. One assume that the fever came from the lung or the pleura. Ther was a cough, which was dry except for occasiona bloody sputum; nothing is said about pus, foul o otherwise. Then there were attacks of definit pleural pain, which changed to an ache and per sisted. Then there was fluid that took a long tim to disappear. At one time the fluid was bloody otherwise it was clear.

So far as the findings by x-ray are concerned there was what was called a thickened pleura, which persisted over a long period of time. The heart was displaced toward the affected side, and it looks as if there was some underlying lung condition, because they describe a partial collapse of the lower lobe.

So far as the heart is concerned there were dyspnea, cyanosis, edema of the ankles, a large liver and fluid in the abdomen. That concludes the story of his symptoms, but along with that we are given a good deal of information. They tell us that he had small cardiac pulsations, a low blood pressure, a low pulse pressure and electrocardiographic findings that, to my knowledge, are consistent with constrictive pericarditis. I do not see how one can get away from such a diagnosis. Except for the pericardial changes, I cannot see how the heart was involved. This man was sixty-five years old. I grant that that is above the average for constrictive pericarditis, but it can happen at that age.

Let us look at the lung films and guess as much as we can about the lung condition.

Dr. Milford D. Schulz: The earliest chest film that I can find is a lateral view taken in 1943, three years before his death.

Dr. King: I think that it shows some involvement in the lung base.

Dr. Schulz: I agree. In the 1944 films you can see collapse of the left lower lobe and some pleural deformity on each side. In those of 1945, there is a newly developed density described as being in the dorsal division of the lower lobe. I wonder, however, if it is not in the basal portion of the upper lobe.

Dr. King: All you can say is that he had partial collapse of the lower lobe and pleural involvement?

Dr. Schulz: Yes. I am looking for calcification in the pericardium, but I do not see any.

Dr. King: These are the films on the last admission, when he came in with what I should say was an acute pneumococcal pneumonia, which I am going to add on top of the old process. He had plenty of Type 3 pneumococci in the throat, not actually in the sputum. Does that not look more like an acute pneumonic process than an infarct?

Dr. Schulz: Yes. If it is an infarct, it is a big one.

Dr. King: Would you be willing to agree that these films are consistent with an acute pneumonia that later spread to the apex of the lung?

Dr. Schulz: Yes; that is what I thought it was when I looked at it.

Dr. King: So far as the pleural process is concerned, I do not know of anything that will give a pleural reaction of this severity and duration and with fluid except tuberculosis. And if one accepts that diagnosis for the pleurisy, one had best accept it also for the pericarditis. The literature tells of constrictive pleuritis, as well as constrictive pericarditis, but there is no evidence of constrictive pleuritis here.

I realize that two pieces of evidence have been given that are against the diagnosis of tuberculosis — a negative guinea-pig inoculation of the chest fluid and a negative biopsy of the pleura. But 35 cc. of fluid is too small an amount for satisfactory examination for tubercle bacilli, especially if one also looks for tumor cells; and although I am not sure of this, I believe that a tuberculous pleurisy may in certain areas show only collagenous tissue, with no definite tubercles. I realize, too, that when pericarditis and pleuritis occur together they are often idiopathic and are never adequately explained. Nevertheless I still hold to the diagnosis of tuberculosis of the pleura and pericardium.

The diagnosis of tuberculosis of the lung is still more difficult to substantiate, but I shall guess that there was tuberculosis in the partially collapsed lobe. It is also possible that at death there was considerable tuberculosis outside this area, but we have no proof of that. Apparently the sputum was not examined for tubercle bacilli — a too frequent oversight. I find that these days the first thought is for the chemical constituents, and sputum examination is often not thought of. Speaking of the chemical findings, the albumin-globulin ratio in this case is consistent with constrictive pericarditis.

Next there is the liver. Did he have cirrhosis? And was it a true Pick's disease — mediastinopericarditic pseudo-cirrhosis of the liver. I suppose that that is what he had. Did he also have chronic peritonitis, which is frequent in these cases? Again, in a long-standing case of this sort I suppose that he did.

When he came to die, I think that he had a Type 3 pneumococcus pneumonia and that he died from a pulmonary embolus. I do not see how we can prove that he had infarcts in the lung, but I believe that he probably did have them.

All of which gives us a great many diagnoses, but I seem forced to take them all — tuberculous constrictive pericarditis, tuberculous pleurisy, pulmonary tuberculosis, pulmonary embolus (fatal), pulmonary infarcts, Type 3 pneumococcus pneumonia and "pseudo-cirrhosis of the liver."

Dr. Edward F. Bland: Is there any mention made of cervical veins?

Dr. King: I do not know why we are not given the venous pressure. It would be a help. Whether I can assume they were normal, I do not know.

I am interested to know if you would make a diagnosis of constrictive pericarditis, Dr. Bland?

Dr. Bland: I do not believe so.

Dr. King: Perhaps you know too much about the case.

Dr. Bland: No.

Dr. Albert J. Stunkard: The patient had no

enlargement of the cervical veins when he was admitted.

DR. KING: He had everything else; I wish he had had that too.

DR. MALLORY: It is obvious that at one period a malignant tumor was strongly considered. Such a possibility unquestionably underlay the biopsy of the pleura.

DR. KING: I grant you that one has to think of malignant tumor in the differential diagnosis, which I did not mention because of time. But the story is too long in duration, and in addition, the patient had a two-year period when he was perfectly well.

CLINICAL DIAGNOSES

Lobar pneumonia.
Carcinoma of lung?
Arteriosclerotic heart disease.
Congestive heart failure.
Pulmonary embolism.
Ventricular fibrillation?

DR. KING'S DIAGNOSES

Tuberculous constrictive pericarditis.
Tuberculous pleurisy.
Pulmonary tuberculosis.
Pneumococcal pneumonia (Type 3).
Pulmonary infarcts.
Pulmonary embolus (fatal).
"Pseudo-cirrhosis of liver."

ANATOMICAL DIAGNOSES

Pericarditis, adhesive.
Pleuritis, fibrous: left chest.
Cirrhosis of liver, toxic.
Hydrothorax, right.
Infarcts of lung, multiple, recent.
Pulmonary emboli, multiple.
Bronchopneumonia, organized.
Osteomyelitis, healed: right fibula.
Pancreatitis, chronic.

PATHOLOGICAL DISCUSSION

DR. MALLORY: Dr. King made a number of diagnoses, and I have to list a number of pathological findings. I do not believe I can tie mine together any more satisfactorily than he has.

There was a constrictive pericarditis. The pericardium was densely fibrous, ranging from 3 to 5 mm. in thickness, with no calcification at any spot. Microscopically the sections showed almost pure collagen, with only the smallest collections of lymphocytes and nothing to suggest tuberculosis. The left pleural cavity was also completely obliterated by old fibrous adhesions, and at the left base, between the lung and diaphragm, this fibrous tissue was 2 to 3 cm. in thickness, being dense and practically cartilaginous in consistence. The right pleural cavity contained a liter of cloudy fluid, probably a terminal accumulation. The lungs showed two distinct types of lesions. There were a large number of pulmonary infarcts — some quite fresh and others slightly older; none of them were very old, however. There were numerous pulmonary emboli to go with them, including a massive embolus, which apparently caused the sudden death. Also scattered irregularly throughout both lungs, but most marked in the right upper lobe, were areas of old completely organized pneumonia. It is quite clear that these were not infarcts, but it is impossible on the basis of the sections to guess how old the process was. There appeared to be nothing active about it in the terminal period; it might have been months or even years old.

Finally, there was a marked cirrhosis of the liver. This was the coarsely nodular type and, to my eye, did not resemble cardiac cirrhosis, which is so often seen in cases of adhesive pericarditis. It was probably a separate lesion of entirely different etiology. Another incidental finding, probably of no significance, was chronic interstitial pancreatitis of moderate severity.

DR. KING: And no tuberculosis?

DR. MALLORY: None anywhere.

DR. ALLAN G. BRAILEY: Do you think that the bloody pleural fluid was due to infarcts at various times?

DR. MALLORY: The fluid was found several years before the final entry, if I remember correctly. The old scars in the lung all look like those of organized pneumonia, not of infarcts. I think that is a distinction we can make with reasonable certainty because the scar of a healed infarct is rather characteristic.

DR. KING: Do these idiopathic processes usually produce as much fever? Have you had cases like this before with persistent temperature?

DR. MALLORY: I do not dare answer that.

DR. BLAND: Did the pulmonary infarcts account for the peaks of temperature?

DR. KING: They might have, I suppose.

DR. MALLORY: It is rather interesting that, despite the cirrhosis of the liver, the numerous pulmonary infarcts did not produce jaundice in the terminal period, as they commonly do with other types of cardiac disease.

DR. BLAND: Were the veins examined?

DR. MALLORY: There were thrombi in the leg veins.

DR. KING: Was this a pneumococcal pneumonia?

DR. MALLORY: There was no acute pneumonia. I believe that all the fresh episodes were due to infarction.

CASE 32102

PRESENTATION OF CASE

A seventy-eight-year-old man entered the hospital complaining of abdominal pain.

During the few months before admission he had occasional bouts of intermittent lower abdominal

pain and was markedly constipated. There was no blood in the stools. Three days before admission, poorly localized abdominal pain again appeared, with pain in the rectum. During this three-day period he had no bowel movements. A few hours before admission he vomited a small amount on three occasions.

The past history revealed that the patient had been constipated for many years, using a laxative daily to obtain a normal bowel movement. For several years he had complained of progressively severer frequency, nocturia and urgency. A year and a half before this admission the prostate was found to be moderately enlarged. A transurethral resection was done, from which he recovered uneventfully.

Physical examination revealed a well developed and nourished man. The skin was warm and moist. The pupils were normal. The heart was not enlarged, and there were no murmurs. There were a few coarse rales at both lung bases. The abdomen was soft, and there was slight tenderness in both lower quadrants. Peristalsis was normal. The prostate was small. The reflexes were normal.

Temperature was 100°F., the pulse 100, and the respirations 25. The blood pressure was 160 systolic, 85 diastolic.

The hemoglobin was 85 per cent. The white-cell count was 14,000. The urine was normal. The nonprotein nitrogen was 31 mg. per 100 cc. The total serum protein was 5.8 gm. per 100 cc., and the chloride 90 milliequiv. per liter. The blood Hinton reaction was negative.

A flat film of the abdomen showed a large amount of gas and retained bowel contents in the large bowel without evidence of obstruction. Several loops of large bowel were dilated. The bones of the lumbar spine showed advanced hypertrophic changes.

A barium enema shortly after admission showed numerous diverticulums of the sigmoid. No spasm was observed fluoroscopically. The bowel wall in this area was irregular. The terminal ileum was not visualized. Shortly after the barium enema had been given, the abdomen was noted to be tense and boardlike, with absent peristalsis, and tenderness became generalized.

About two hours later a plain film showed the greater portion of the barium to be retained in the large bowel. A film with the patient standing showed no evidence of air beneath the diaphragm.

An immediate exploratory laparotomy was done.

Differential Diagnosis

Dr. Fiorindo A. Simeone: This is the case of a seventy-eight-year-old man who had a lesion which caused partial obstruction of the large intestine and developed a complication necessitating an emergency operation. There is no supporting evidence that the final episode of abdominal spasm and tenderness could have been due to "abdominal apoplexy" or to volvulus and strangulation. These possibilities are therefore dismissed, and I believe that it is reasonable to explain the present illness and the final episode on the basis of a single lesion.

One has to decide whether the lesion causing partial obstruction was intrinsic or extrinsic relative to the bowel. The patient had a history of mild urinary-tract obstruction, which might have been due to cancer of the prostate, but this was apparently cured by transurethral resection and there is no evidence of recurrence. Rectal examination on entry showed a normal prostate, and nothing is recorded to suggest a pelvic mass that could have caused obstruction of the large intestine from extrinsic pressure. The roentgenograph apparently did not suggest adynamic ileus, and the negative Hinton reaction rules out the adynamic ileus occasionally seen in tabes dorsalis.

We are thus left with a lesion that was probably intrinsic in the large intestine and certainly interfered mechanically with the normal emptying of the bowel. Five important lesions are to be considered in the differential diagnosis of large-bowel obstruction in a man of seventy-eight — malignant tumor, diverticulitis, hernia, volvulus and appendiceal abscess. The lack of signs in the right lower quadrant, the failure to demonstrate a mass and the localization of the nonspastic lesion in the sigmoid by roentgenograph are sufficient to rule out appendicitis with abscess formation. Physical examination and the x-ray findings rule out hernia and volvulus. It remains, therefore, to distinguish between diverticulitis and carcinoma of the sigmoid. Tumors of mesodermal origin in the wall of the large bowel are not considered because they are rare and because they do not lead to perforation without involving the mucosa. Granulomas — tuberculosis and actinomycosis — are also rare in this region and need not be discussed in the absence of more evidence suggesting them. Without more definite evidence concerning the nature of the mucosa in the region of the lesion, one can merely marshal the evidence and then make as good a guess as one can regarding the differentiation of carcinoma and diverticulitis.

The long history of constipation is characteristic of diverticulitis and diverticulosis but does not help us any more than does the demonstration of diverticulums by roentgenograph. In fact, if the patient used daily catharsis for years, it is difficult to be sure he was really constipated. Some change took place in the bowel during the three days before admission, however, for then there were no bowel movements. I assume that the change was inflammatory.

The patient was seventy-eight years old. The ages most frequently encountered in diverticulitis are in the decade between fifty and sixty. When these figures are corrected for the incidence of ages in the general population, however, diverticulitis

becomes about as common in occurrence in the older age groups as it is in the fifty-to-sixty group.

Diverticulitis is more frequent among males than among females, but this point is of little help in differential diagnosis.

Lack of blood in the stools is certainly in favor of diverticulitis, as opposed to carcinoma of the sigmoid. It is true, of course, that some patients with carcinoma do not have blood in the stools and that 10 to 20 per cent of those with diverticulitis do show this sign. Nevertheless, the lack of blood in the stools can be interpreted as strongly in favor of diverticulitis.

A mass is felt more frequently with diverticulitis than with carcinoma. No definite statement is made that no mass was palpated, but it can be assumed that none was found. Absence of a mass, however, is not conclusive evidence against diverticulitis.

Slight tenderness in both lower quadrants is consistent with diverticulitis. It is equally consistent, although less often elicited, with carcinoma complicated by penetration or perforation. Such a carcinoma, however, should have been demonstrated by obvious mucosal changes in the roentgenogram.

The fever and leukocytosis suggest an inflammatory reaction. Although these signs favor diverticulitis, they do not exclude carcinoma, which, as already mentioned, may have an inflammatory reaction about it.

Hypoproteinemia and hypochloremia are recorded as isolated occasions. These observations can be assumed to be accurate and are not extraordinary in an old man who has had chronic intestinal obstruction with recent vomiting. The patient may not actually have been as well nourished as he seemed.

The roentgenographic finding of diverticulums is strongly in favor of diverticulitis as the cause of the patient's symptoms. It does not exclude carcinoma, however, for cancer may coexist with diverticulitis and diverticulosis. The irregularity of the bowel wall is typical of diverticulitis. If we are justified in assuming that no abnormality was detected in the mucosa itself, this is strong evidence against carcinoma. The lack of spasm is against diverticulitis but does not rule it out. The degree to which the lumen was narrowed, if at all, is not stated. If there was little narrowing, the finding would favor diverticulitis.

On weighing all the evidence, we find more in favor of diverticulitis than of carcinoma. The reference of pain about the rectum is more consistent with diverticulitis than with uncomplicated carcinoma. The final episode of generalized tenderness and boardlike spasm in the abdomen indicates a generalized peritonitis, which followed shortly after the administration of a barium enema. This is more readily explained on the basis of diverticulitis than on that of carcinoma. It may be assumed that the manipulation or the pressure de veloped when unsuccessfully attempting to expe the enema caused perforation of a gangrenou diverticulum. This resulted in a spreading peri tonitis. Such occurrences are rare but have bee described. The same picture could have been pro duced by the rupture of an abscess about the are involved in the diverticulitis. This is suggested b the fact that no gas was found beneath the dia phragm by x-ray examination when the patien was examined in the standing position. Absence o demonstrable air beneath the diaphragm, howeve does not rule out perforation of the bowel at th site of a diverticulitis. Perforation of a malignan lesion of the colon is not infrequent during proc toscopy or sigmoidoscopy, but I am not aware o such a complication following a barium enema.

The question arises whether perforation may hav occurred in the bowel at a site other than that o the lesion itself. Rupture of the large intestine a the result of obstruction in the colon usually occur in the cecum. Had this taken place in this patient air beneath the diaphragm would have been ex pected and there should have been evidence o severer bowel obstruction beforehand than there apparently was.

In summary, the patient's long history of con stipation, the low abdominal pain and tenderness the pain in the rectum, the mild fever and leuko cytosis, the evidence of incomplete large-bowe obstruction by history and roentgenograph, the x-ray evidence of a partially obstructing lesion in the sigmoid with diverticulums nearby and with out obvious destruction of the mucosa and the ab sence of blood in the stools suggest a diagnosis o diverticulitis. The episode that led to emergency surgery was a spreading peritonitis as the result o perforation of an involved diverticulum.

Clinical Diagnoses

Peritonitis from perforated viscus.
Hypertension.
Arteriosclerosis.

Dr. Simeone's Diagnoses

Diverticulitis of colon, with perforation and generalized peritonitis.
Diverticulosis of colon.

Anatomical Diagnoses

Adenocarcinoma of rectosigmoid, with perforation. General peritonitis.
Operative wound: transverse colostomy.
Cardiac hypertrophy, hypertensive type.
Arteriosclerosis of coronary arteries, moderate.
Arteriosclerosis of aorta, severe.
Pulmonary atelectasis.
Diverticulosis of colon.
Prostatectomy, old, transurethral.

Pathological Discussion

Dr. Tracy B. Mallory: Clinical discussion on

the wards essentially paralleled Dr. Simeone's reasoning. Diverticulitis was considered the most probable diagnosis, and the sudden episode was interpreted as a perforation. An exploratory laparotomy was performed by Dr. J. G. Scannell. He found a diffuse purulent peritonitis, most marked in the pelvis. The appendix was normal. A mass was found at the rectosigmoid junction that was thought to be inflammatory and secondary to a perforated diverticulum. A transverse colostomy was done, and the patient was put on the usual regime for peritonitis. His general condition steadily deteriorated, and he died on the fifth day after operation.

The post-mortem examination showed a severe diffuse peritonitis that involved all parts of the peritoneal cavity without any localized collection to suggest abscess formation. A mass could be felt at the junction of the sigmoid and rectum that constricted the bowel, reducing the lumen to a diameter of 2 cm. On opening the bowel a characteristic ulcerative carcinoma was found, with a perforation in the center of the area of ulceration. The perforation was small, and no free fecal material was found in the peritoneal cavity. Although diverticulums were present throughout the bowel none showed evidence of inflammation.

The other findings at autopsy were for the most part coincidental. There was massive collapse of the lower lobe of the right lung. The heart showed a hypertensive hypertrophy, and although the coronary arteries were sclerotic and narrowed, there was no evidence of infarction. The gall bladder was large and contained numerous large stones. The major portion of the prostate had been removed, and there was comparatively little regeneration.

Microscopical examination proved the tumor in the bowel to be a well differentiated adenocarcinoma. There were no implantations or metastases.

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CARDIAC MURMURS

A letter published elsewhere in this issue of the *Journal* criticizes the stand taken in a recent paper* regarding the significance of apical systolic murmurs. Undoubtedly, in the interpretation of heart murmurs the pendulum swung from one extreme to the other thirty years ago, when James Mackenzie urged the medical profession to disregard auscultatory cardiac findings, provided there was an adequate state of the circulatory function. That advice served a useful purpose — it did help win World War I by allowing many able-bodied men with heart murmurs to serve in the British forces. But the interpretation was too literal, and for many years British medicine suffered from its indiscriminate acceptance. One cardiologist states that as long as ten years after the end of World War I he found cases of clear-cut and clinically significant aortic stenosis on the wards of Thomas Lewis that had been undiagnosed because of the scorn of stethoscopic findings. In other words, the pendulum was still at its extreme. During the last two decades, however, it has become the experience of most cardiologists who went through the earlier period that this casual attitude is not only unscientific but actually dangerous. There are three reasons for this.

In the first place, valvular disease produces a strain on the heart muscle; at times it is slight or moderate, but sometimes it is considerable, even though cardiac function and reserve may, for the moment, be adequate. In almost all cases trouble looms up ahead, and the more careful the patient is to avoid unnecessary strain, the longer is the postponement of trouble. Of course, the development of a crippling neurosis or valetudinarianism should be avoided, but so too should the unnecessary early induction of acute pulmonary edema by a disregard of the considerable strain resulting from a previously symptomless but easily diagnosed aortic stenosis. There is a happy mean in the conduct of such a person's life.

In the second place, a healthy young person with chronic valvular disease is a candidate for recurrent rheumatic fever if exposed to infection by the hemolytic streptococcus, as experience in World War II has so clearly demonstrated. Such a person obviously deserves some protection.

Finally, it is true that one of the most infallible guides to the presence of active rheumatic infection in a doubtful case is the development of a heart murmur *under observation*, especially the controversial apical systolic murmur not explained by anemia or other factors but due to left ventricular dilatation secondary to acute rheumatic myocarditis.

Any moderate or loud apical or aortic systolic murmur in a person *at rest* is best regarded as abnormal unless proved to the contrary. The cause may, of course, be extracardiac, but it should be looked for and diagnosed, whether intracardiac or extracardiac.

No brief should be held for the exaggeration of

*Wolfson, I. N. Clinical and laboratory studies of 91 workers with apical systolic murmurs. *New Eng. J. Med.* 233:757-761, 1945.

the importance of heart murmurs, in fact their neglect is the lesser of the two evils, but it is possible with care and experience to avoid both extreme points of view and to develop the use of the stethoscope as an extremely valuable medical tool in the diagnosis of heart disease.

SCHOOL-HEALTH POLICIES

School health in its various aspects and the impact of school medical services on health education have received nearly their share of attention in the last decade. It is up to those who are interested in it, however, to see that school health does not fall too far into the category of Mark Twain's New England weather, about which so many have talked and so few have attempted any practical reforms.

The Strayer Report on the Boston Public Schools, published in 1944 in its various volumes, contained recommendations on school lunches and on physical and health education that were considered by a committee of the Boston Health League and, in the main, favorably reported on. The Division of Child Hygiene of the Massachusetts Department of Public Health has developed excellent procedures to be used as guides in the towns and cities of Massachusetts outside Boston; studies have been made jointly by the State Department of Health and the State Department of Education; and many other wise pronouncements the Nation over are available for those who wish to profit by them.

If there is any real reason why the country's schools do not have and do not follow the best and most progressive health policies, it is because of a public that is bent on other forms of whoopee or because of the inertia of municipal politics when applied to academic problems.

The American Academy of Pediatrics interested itself in the problem of school health soon after its organization in 1931 and formed a committee to study the subject. It has published various reports, including a statement "Suggested School-Health Policies," produced jointly with the Child Hygiene Section of the American Public Health Association. A revision of this report has been currently made public by the National Conference for Co-operation in Health Education,* and the pamphlet includes about all the advice that can be given to anyone interested in establishing school-health policies in any locality.

Particularly important with respect to these modern school-health policies is the necessity of raising school health and health education to the position of dignity and importance that belongs to them. Teachers should be prepared at teachers' colleges to assume their share of the health program; school nursing should be on a career level; the branch of public health that deals with school health and health education should be expected to furnish careers for an adequate number of trained physicians. The school should participate in community health education, and conversely, the community should interest itself in the health policies of its schools. Inside the school, the health council should be a representative body including students and parents, and this council should co-ordinate with the central school-health council and the community health council.

As with so many of life's obstacles, the problem lies not so much in finding a way to follow as in finding the will to follow the way.

MASSACHUSETTS MEDICAL SOCIETY

REVIEW LECTURE COURSE

The following is the detailed program of the Review Lecture Course from March 18 to April 15, inclusive.

March 18. GASTROINTESTINAL DISEASES. *Chairmen*: Sara M. Jordan and Franklin W. White.

5:00–5:30. Diagnosis of Cancer of the Digestive Tract. Franz J. Ingelfinger: assistant professor of medicine, Boston University School of Medicine; gastroenterologist, Massachusetts Memorial Hospitals.

5:30–6:00. Functional Diseases of the Digestive Tract. Sara M. Jordan: chief, Department of Gastroenterology, Lahey Clinic; physician, New England Baptist and Deaconess hospitals.

6:00–6:30. Ulcerative Colitis. Isaac R. Jankelson: assistant professor of medicine, Tufts College Medical School; junior visiting physician, Boston City Hospital.

7:30–8:00. Epidemiology of Amebiasis and the Dysenteries. Vlado A. Getting: Commissioner of Public Health, Commonwealth of Massachusetts.

8:00–8:30. Diseases of the Small Intestine. Katherine S. Andrews: surgeon, New England Hospital for Women and Children.

8:30–9:00. Some Proctologic Problems. E. Parker Hayden: assistant in surgery, Harvard Medical School; assistant surgeon, Massachusetts General Hospital.

*The American Academy of Pediatrics and the American Public Health Association. *Suggested School-Health Policies.* Second edition revised. 57 pp. 1945.

March 20. DISEASES OF THE LIVER, GALL BLADDER AND PANCREAS. *Chairmen*: Chester M. Jones and Leland S. McKittrick.

3:00–3:45. Epidemiology of Hepatitis. Roy F. Feemster: director, Division of Local Health Administration, Massachusetts Department of Public Health.

3:45–4:30. Chronic Hepatic Disorders. Chester M. Jones: clinical professor of medicine, Harvard Medical School; physician, Massachusetts General Hospital.

4:30–5:15. Surgery of the Biliary Tract. Charles G. Mixter: surgeon-in-chief, Beth Israel Hospital.

5:15–6:00. Diagnosis and Treatment of Carcinoma of the Pancreas. Richard B. Cattell: surgeon, Lahey Clinic; surgeon, New England Deaconess and Baptist hospitals.

March 25. SURGERY. *Chairmen*: Edward D. Churchill and John W. Spellman.

5:00–5:30. Progress Report. F. D. Moore: instructor in surgery, Harvard Medical School; assistant in surgery, Massachusetts General Hospital.

5:30–6:00. Military Surgery. E. D. Churchill: John Homans Professor of Surgery, Harvard Medical School; chief of West Surgical Service, Massachusetts General Hospital.

6:00–6:30. Shock and Resuscitation. J. E. Dunphy: associate in surgery, Harvard Medical School; senior associate in surgery, Peter Bent Brigham Hospital; visiting surgeon, St. Elizabeth's Hospital.

7:30–8:15. Choice of the Anesthetic Agent. Morris J. Nicholson: anesthesiologist, Lahey Clinic and New England Baptist and Deaconess hospitals.

8:15–9:00. Head Injury. Donald Munro: associate professor of neurosurgery, Boston University School of Medicine; assistant professor of neurological surgery, Harvard Medical School; surgeon-in-chief and head of Department of Neurosurgery, Boston City Hospital.

March 27. SURGERY. *Chairmen*: Edward D. Churchill and John W. Spellman.

3:00–3:45. Thermal Burns. Charles C. Lund: assistant professor of surgery, Harvard Medical School; visiting surgeon, Boston City Hospital.

3:45–4:30. Phlebitis, Thrombosis and Embolism. Claude E. Welch: assistant in surgery, Harvard Medical School; assistant visiting surgeon, Massachusetts General Hospital.

ARTHRITIS. *Chairmen*: Marian W. Ropes and Howard K. Thompson.

4:30–5:15. Problems in the Treatment of Various Types of Chronic Arthritis. Nathan Sidel: chief of Arthritis Clinic, Beth Israel Hospital; assistant visiting physician, Boston City Hospital; assistant professor of medicine, Tufts College Medical School.

5:15–6:00. Newer Concepts in the Management of Rheumatoid Arthritis With Special Reference to Gold Salts. Theodore Bayles: visiting physician, Robert Breck Brigham Hospital; junior associate in medicine, Peter Bent Brigham Hospital.

April 1. ARTHRITIS. *Chairmen*: Marian W. Ropes and Howard K. Thompson.

5:00–5:45. Orthopedic Care of the Chronic Arthritic. John Kuhns: instructor in orthopedic surgery, Harvard Medical School; visiting orthopedic surgeon, Robert Breck Brigham and Infants' hospitals.

5:45–6:30. Physiotherapy in the Treatment of Arthriti Arthur Watkins: chief of physical medicine, Massachusett General Hospital; associate in medicine, Harvard Medica School.

DISEASES OF THE BONES AND JOINTS. *Chairmen*: Frank R Ober and William A. Rogers.

7:30–8:15. Lame Backs. William A. Rogers: instructor in orthopedic surgery, Harvard Medical School; visiting orthopedic surgeon, Massachusetts General Hospital.

8:15–9:00. Lame Shoulders. Frank R. Ober: John B. an Buckminster Brown Clinical Professor of Orthopedi Surgery, Harvard Medical School; orthopedic surgeon Peter Bent Brigham Hospital; chief of Orthopedic Depart ment, Children's Hospital.

The Journal lacks copies of the December 6, 1945 and January 3, 1946, issues. If any subscribers who do not bind their copies have the above-mentioned issues on hand, the Journal will gladly pay 15 cents for each copy left at or mailed to its office (8 Fenway, Boston 15).

April 3. FRACTURES. *Chairmen*: Frank R. Ober an William A. Rogers.

3:00–3:45. Colles Fracture. Henry C. Marble: assistan in surgery, Harvard Medical School; visiting surgeon Massachusetts General Hospital; surgeon-in-chief, Chelse Memorial Hospital.

3:45–4:30. Fractures of the Elbow. Carroll B. Larson assistant in orthopedic surgery, Massachusetts Genera Hospital; assistant in surgery, Harvard Medical School.

NEUROSURGERY OF CHILDHOOD. *Chairmen*: Franc D Ingraham and James B. Campbell.

4:30–6:00. Congenital Anomalies, Injuries, Infections Intracranial and Intraspinal Tumors, Jacksonian Epilepsy and Lead Encephalitis. Franc D. Ingraham: surgeon-in chief, Children's Hospital; assistant professor of surgery Harvard Medical School. James B. Campbell: junior attending surgeon in neurosurgery, Children's Hospital assistant in surgery, Harvard Medical School.

April 8. KIDNEY AND GENITOURINARY DISEASES. *Chairmen* Laurence B. Ellis and James P. O'Hare.

5:00–6:30. Glomerular Nephritis and Nephrosis: Progress in etiology, pathology, physiology and clinical management Stanley E. Bradley: instructor in medicine, Boston Uni versity School of Medicine; assistant physician, Evans Memorial, Massachusetts Memorial Hospitals. W. Richard Ohler: assistant professor of medicine, Harvard Medical School; chief, Second Medical Service, Boston City Hospital.

7:30–8:15. Urinary-Tract Infections. E. Granville Crabtree: urologist, New England Baptist, Newton and Beth Israel hospitals.

8:15–9:00. The Female Bladder. Samuel N. Vose: professor of genitourinary surgery, Boston University School of Medicine; urologist, Massachusetts Memorial Hospitals.

April 10. PEDIATRICS. *Chairmen*: James M. Baty and Joseph Garland.

3:00–3:45. Care and Feeding of Newborn. Stewart H Clifford: pediatrician, Boston Lying-in Hospital; assistant visiting physician, Children's Hospital.

3:45–4:30. Surgical Procedures in Early Months of Life Robert Gross: assistant professor of surgery, Harvard Medical School.

4:30–5:15. Immunization Procedures. Allan M. Butler assistant professor of pediatrics, Harvard Medical School chief, Children's Medical Department, Massachusetts General Hospital.

5:15–6:00. Rheumatic Fever. T. Duckett Jones: director of research, House of the Good Samaritan.

April 15. DERMATOLOGY. *Chairmen*: John G. Downing and C. Guy Lane.

5:00–6:30. Symposium on Dermatitis, Eczema and Allergy. John G. Downing: professor of dermatology and syphilology, Tufts College Medical School; professor of dermatology, Boston University School of Medicine; dermatologist-in-chief, Massachusetts Memorial, Boston City and St. Elizabeth's hospitals. C. Guy Lane: clinical professor of dermatology, Harvard Medical School; chief, Dermatologic Department, Massachusetts General Hospital. Francis M. Rackemann: lecturer in medicine, Harvard Medical School; physician, Massachusetts General Hospital. John Fromer: director, Department of Allergy and Dermatology, Lahey Clinic.

7:30–7:50. Treatment and Diagnosis of Cutaneous Disturbances of Nutritional Origin and Endocrine Dysfunction. G. Marshall Crawford: instructor of dermatology, Harvard Medical School; assistant dermatologist, Dermatologic Department, Massachusetts General Hospital.

7:15–8:15. Advances in the Treatment of Pyogenic Infections, Psoriasis and Allied Diseases. Bernard Appel: associate professor of dermatology, Tufts College Medical School; assistant dermatologist, Boston City Hospital.

8:15–8:35. Management of Fungous and Parasitic Infections of the Skin. Jacob H. Swartz: assistant professor of dermatology, Harvard Medical School; dermatologist, Dermatologic Department, Massachusetts General Hospital.

8:35–9:00. Hypertrophies, Atrophies, and Neoplasms of the Skin. Francis M. Thurmon: assistant professor of dermatology, Tufts College Medical School; physician-in chief, Clinic of Dermatology and Syphilology, Boston Dispensary.

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OLSON, JOHN H., Colrain.
College of Physicians and Surgeons, Boston, 1936. Sponsor: Harry L. Craft, Ashfield.

Harry L. Craft, *Secretary*
Ashfield

HAMPDEN DISTRICT

APPLEFORD, GEORGE B., Springfield Hospital, Springfield.
Tufts College Medical School, 1941.

ASHE, ALLAN M., 262 Riverdale Street, West Springfield.
College of Physicians and Surgeons, Boston, 1937. Sponsor: Alfred M. Glickman, 1938 Main Street, Springfield.

BILSKI, THEODORE D., 12 Pleasant Street, Westfield.
University of Halle, 1922. Sponsor: Archibald J. Douglas, 30 Court Street, Westfield.

CONWAY, RAYMOND F., 2068 Northampton Street, Holyoke.
Boston University School of Medicine, 1941.

CURTIS, SPRAGUE, 24 Bevier Street, Springfield.
Tufts College Medical School, 1938.

DAVID, LEO J., 419 Sumner Avenue, Springfield.
Berlin Medical School, 1919. Sponsor: Edward Katz, 10 Chestnut Street, Springfield.

DERRICK, CHARLES C., 58 Manor Road, Springfield.
Rush Medical College, University of Chicago, 1939.

EGNATZ, NICHOLAS, Westfield State Sanatorium, Westfield.
Indiana University School of Medicine, 1943.

KIBBE, MILTON H., 67 Harwich Road, West Springfield.
Tufts College Medical School, 1938.

KNOX, BARRON D., 1157 Carew Street, Springfield.
Tufts College Medical School, 1937.

MINER, THEODORE R., 960 Longmeadow Street, Longmeadow.
New York University and Bellevue Hospital Medical College, 1927.

MURRAY, MARTIN B., 952 State Street, Springfield.
New York University School of Medicine, 1937.

SEARS, WARREN H., 17 Kenwood Terrace, Springfield.
Johns Hopkins University School of Medicine, 1933.

STEVENS, JOSEPH E., 254 Rogers Avenue, West Springfield.
Middlesex University School of Medicine, 1928. Sponsor: George B. Corcoran, 84 Park Street, West Springfield.

VASSOS, GEORGE A., JR., 26 Mulberry Street, Springfield.
Cornell University Medical College, 1939.

George C. Steele, *Secretary*
39 Upper Church Street, West Springfield

MIDDLESEX EAST DISTRICT

CASS, VICTORIA M., 20 Ridgefield Road, Winchester.
Tufts College Medical School, 1942.

LEWIS, DONALD K., 8 Glen Gary, Winchester.
University of Michigan Medical School, 1940.

TOPPENBERG, DAVID R., New England Sanitarium and Hospital, Melrose.
College of Medical Evangelists, 1938.

WESTERMEYER, MARION W., New England Sanitarium and Hospital, Melrose.
College of Medical Evangelists, 1938.

Roy W. Layton, *Secretary*
8 Porter Street, Melrose

MIDDLESEX NORTH DISTRICT

GILMORE, RAY S., 150 Nesmith Street, Lowell.
Tufts College Medical School, 1941.

MARTINSON, MELVIN S., 468 Main Street, Acton.
Tufts College Medical School, 1939.

MCCARTHY, THOMAS F., 46 Holyrood Avenue, Lowell.
Harvard Medical School, 1937.

MEAD, SEDGWICK, Tahattawan Road, Littleton.
Harvard Medical School, 1938.

ROSENBAUM, EMIL E., 617 Westford Street, Lowell.
University of Berlin, 1918. Sponsor: Archibald R. Gardner, 16 Shattuck Street, Lowell.

VERNUM, JOSEPH B., 858 Chelmsford Street, Lowell.
Tufts College Medical School, 1941.

Brendan D. Leahey, *Secretary*
9 Central Street, Lowell

MIDDLESEX SOUTH DISTRICT

ABRAMS, SIDNEY M., 606 Main Street, Malden.
Middlesex University School of Medicine, 1939. Sponsor: Eugene G. Auld, 568 Main Street, Malden.

ANDALORO, VINCENT A., 6 Crescent Street, Everett.
Middlesex University School of Medicine, 1938. Sponsor: Harris Bass, 351 Broadway, Everett.

APSHAGA, BRONIE J. P., 490 Western Avenue, Brighton.
Tufts College Medical School, 1942.

BLANK, JOSEPH G., 123 Summit Street, Waltham.
Middlesex University School of Medicine, 1939. Sponsor: Lee G. Kendall, Belknap Road, Framingham.

BLESOFF, BENJAMIN, 326 Highland Avenue, Somerville.
Middlesex University School of Medicine, 1937. Sponsor: Charles O. Nauen, 773 Broadway, Somerville.

CARADONNA, MATTEO, 132 Chelsea Street, Everett.
Middlesex University School of Medicine, 1938. Sponsor: Frank Mirabello, 332 Broadway, Everett.

COZZA, LAWRENCE F., 20A Bellevue Street, Medford.
Tufts College Medical School, 1943.

CROISSANT, EDWARD D., 315 Belmont Street, Belmont.
Middlesex University School of Medicine, 1940. Sponsor: Max Ritvo, Newton; office 416 Marlboro Street, Boston.

ELLIS, DANIEL S., 68 Grozier Road, Cambridge.
Harvard Medical School, 1939.

FISHER, ROBERT M., Waltham Hospital, Waltham.
Columbia University College of Physicians and Surgeons, 1939.

HAWN, CLINTON V. Z., 37 Kirkland Street, Cambridge.
Harvard Medical School, 1941.

KALIKA, KARL, 1638 Commonwealth Avenue, Brighton.
Midwest Medical College, 1934. Sponsor: Arthur M. Simmons, 1284 Commonwealth Avenue, Allston.

LANDSTEINER, ERNEST K., 26 Walnut Hill Road, Newton Centre.
Harvard Medical School, 1941.

LEWIS, HERBERT D., 56 Eddy Street, West Newton.
Harvard Medical School, 1942.

MANDRACHIA, ALFONSO C., 9 Wolcott Street, Everett.
Middlesex University School of Medicine, 1939. Sponsor: Frank Mirabello, 332 Broadway, Everett.

MCCARTHY, ALLAN J., 1173 Massachusetts Avenue, Arlington.
Middlesex University School of Medicine, 1935. Sponsor: Edward W. Feeley, 15 Medford Street, Arlington.

MCDONOUGH, FRANCIS E., 164 Fairway Drive, West Newton.
University of Wisconsin Medical School, 1934.

MEROLA, JOSEPH F., 197 High Street, Waltham.
Middlesex University School of Medicine, 1935. Sponsor: Irving W. Fraim, 826 Main Street, Waltham.

MORAN, RICHARD J., 6 Colonial Drive, Arlington.
Tufts College Medical School, 1940.

PAGLIUCA, GERALD F., 97 Dartmouth Street, Medford.
College of Physicians and Surgeons, Boston, 1934. Sponsor: John W. Gahan, 27 Washington Street, Medford.

PROCHNIK, JAMES J., 243 Harvard Avenue, Allston.
University of Vienna, 1919. Sponsor: Joseph E. F. Riseman, 45 Bay State Road, Boston.

RAWSON, RULON W., 84A Ellery Street, Cambridge.
Northwestern University Medical School, 1938.

RICCI, ALPINE L., 807 Salem Street, Malden.
Middlesex University School of Medicine, 1937. Sponsor: William T. Moriarty. 65 Elm Street, Malden.

SEWALL, EDGAR F., Jr., 166 Sycamore Street, Somerville.
Tufts College Medical School, 1943.

SHORT, LEONARD V., 1433 Massachusetts Avenue, Lexington.
Middlesex University School of Medicine, 1939. Sponsor: Winthrop W. Harrington, 1900 Massachusetts Avenue, Lexington.

SKORNIK, NATHAN H., 323 Langley Road, Newton Centre.
Middlesex University School of Medicine, 1940. Sponsor: Stephen Rushmore, 95 Dudley Road, Newton Centre.

STRASSMANN, GEORGE S., Metropolitan State Hospital, Waltham.
University of Heidelberg, 1913. Sponsor: William C. Gaebler, Metropolitan State Hospital, Waltham.

STURDEVANT, CHARLES L., 32 Blake Street, Newtonville.
University of Nebraska College of Medicine, 1936.

SWEENEY, GUY R., 10 Turell Road, Medford.
Middlesex University School of Medicine, 1936. Sponsor: Paul J. D. Haley, 333 Winthrop Street, Medford.

TEDESCHI, PASQUALE R., 270 Pearl Street, Newton.
Middlesex University School of Medicine, 1939. Sponsor: Sidney Derow, 925 Center Street, Newton Centre.

WARE, PAUL F., 116 Pond Brook Road, Chestnut Hill.
Harvard Medical School, 1943.

WEINSTEIN, LOUIS, 310 Allston Street, Brighton.
Boston University School of Medicine, 1943.

WISE, CHARLES S., 410 Memorial Drive, Cambridge.
New York University College of Medicine, 1939.

Alexander A. Levi, *Secretary*
481 Beacon Street, Boston

NORFOLK DISTRICT

ANDERSON, FRED A., 61 Hastings Street, West Roxbury.
Boston University School of Medicine, 1940.

AUSTEN, GEORGE, Jr., 21 Hawthorn Road, Brookline.
Harvard Medical School, 1934.

BERMAN, SAMUEL S., 257 Warren Street, Roxbury.
Middlesex University School of Medicine, 1935. Sponsor: Hyman B. Friedman, 42 Ogden Road, West Roxbury.

BROOKS, EUGENE F., Wrentham.
Middlesex University School of Medicine, 1939. Sponsor: Karl V. Quinn, Wrentham State School, Wrentham.

CURRENS, JAMES H., 67 Crowninshield Road, Brookline.
Duke University School of Medicine, 1938.

DEMINICO, LUIGI B., 2 Cedarwood Road, Jamaica Plain.
Tufts College Medical School, 1937.

DYER, EDWARD C., 10 Browne Street, Brookline.
Harvard Medical School, 1939.

FAY, THOMAS F., 9 Regina Road, Dorchester.
Georgetown University School of Medicine, 1933.

FRIEDLAND, FRITZ, 53 Westbourne Terrace, Brookline.
University of Berlin, 1934. Sponsor: Georg Lepehne, 30 Summit Avenue, Brookline.

GOLDBERG, GEORGE, 138 Tappan Street, Brookline.
Middlesex University School of Medicine, 1937. Sponsor: Benjamin F. Bornstein, Chestnut Hill; office, 370 Commonwealth Avenue, Boston.

GRENDAL, MICHAEL F., 945 South Street, Roslindale.
Jefferson Medical College, 1942.

HAM, DONALD P., 12 Rosewood Street, Mattapan.
Boston University School of Medicine, 1938.

HOGAN, EDWARD N., 6 Lyndhurst Street, Dorchester.
Georgetown University School of Medicine, 1936.

ISENSTEIN, CHARLES, 57 Lawton Street, Brookline.
Tufts College Medical School, 1939.

KLEIN, LEONARD M., 145 Townsend Street, Roxbury.
Tufts College Medical School, 1940.

LAMPNER, HAROLD, 293 Mason Terrace, Brookline.
Middlesex University School of Medicine, 1937. Sponsor: Gabriel H. Shohet, 103 Columbia Road, Dorchester.

LANE, GEORGE M., 21 Alton Court, Brookline.
College of Physicians and Surgeons, Boston, 1944. Sponsor: William Dameshek, 38 Willow Crescent, Brookline.

LEARY, HOWARD L., 41 Pond Street, Jamaica Plain.
Tufts College Medical School, 1943.

LEVINE, HAROLD D., 108 Longwood Avenue, Brookline.
Harvard Medical School, 1932.

LITTON, NINA, 30 Stearns Road, Brookline.
University of Naples, 1938. Sponsor: C. Wesley Sewall, 135 Bellevue Street, West Roxbury.

MEYERS, MARVIN T., Station Hospital, Camp MacKall, North Carolina (former residence: Jamaica Plain)
Kansas City University of Physicians and Surgeons, 1931. Sponsor: Abram I. Cohen, 491 Commonwealth Avenue, Boston.

PARTON, GEORGE P., Jr., 153 Brook Street, Wellesley.
Middlesex University School of Medicine, 1940. Sponsor: Daniel B. Coleman, 605 Washington Street, Wellesley.

REGAN, FRANCIS C., 7 Hickory Road, Wellesley.
University of Rochester School of Medicine, 1936.

ROBINSON, CHARLES, 56 Columbia Road, Dorchester.
College of Physicians and Surgeons, Boston, 1938. Sponsor: Hyman B. Goldberg, 2 Pasadena Road, Dorchester.

ROSEN, DANIEL, 66 Columbia Road, Roxbury.
University of Vienna, 1936. Sponsor: Hyman B. Goldberg, 2 Pasadena Road, Roxbury.

ROSS, FREDERICK P., 629 Chestnut Hill Avenue, Brookline.
Harvard Medical School, 1939.

SAFFRAN, IRVING, 21 Evelyn Street, Mattapan.
Middlesex University School of Medicine, 1937. Sponsor: Samuel L. Marnoy, Brookline; office, 311 Commonwealth Avenue, Boston.

SORKIN, JOSEPH J., 943 Morton Street, Dorchester.
Chicago College of Medicine and Surgery, 1916.

SCHWARTZ, ROBERT, 1017 River Street, Hyde Park.
Middlesex University School of Medicine, 1939. Sponsor: Albert Ehrenfried, Brookline; office, 520 Beacon Street, Boston.

THORNTON, P. JOHN S., 300 South Street, Brookline.
University of Lausanne, 1934. Sponsor: J. Peter Thornton, 300 South Street, Brookline.

TURANYI, BELA, 15 Virginia Street, Dorchester.
Royal Hungarian Elisabeth University, 1924. Sponsor: James J. Lynch, 801 Columbia Road, Dorchester.

WASKOW, ELEANOR A., 368 Longwood Avenue, Boston.
University of Wisconsin Medical School, 1941.

WILSON, WALTER E., JR., 12 Griggs Terrace, Brookline.
Emory University School of Medicine, 1939.

WOOD, WILLIAM B., 107 Anawan Avenue, West Roxbury.
Tufts College Medical School, 1916.

Basil E. Barton, *Secretary*
10 Richwood Street, West Roxbury

NORFOLK SOUTH DISTRICT

DAVIS, ALBERT, 191 Sea Street, Quincy.
Middlesex University School of Medicine, 1936. Sponsor: William S. Altman, 32 Spear Street, Quincy.

FROGEL, REUBEN H., 170 Quincy Avenue, East Braintree.
Middlesex University School of Medicine, 1933. Sponsor: William S. Altman, 32 Spear Street, Quincy.

Ebenezer K. Jenkins, *Secretary*
Norfolk County Hospital, South Braintree

PLYMOUTH DISTRICT

CASTLETON, HERBERT E., 28 Webster Street, Rockland.
Middlesex University School of Medicine, 1934. Sponsor: Joseph H. Dunn, 321 Union Street, Rockland.

LEBARON, FRANCIS E., 27 Leyden Street, Plymouth.
Middlesex University School of Medicine, 1935. Sponsor: William E. Curtin, 272 Court Street, Plymouth.

NOONAN, JOHN P., 15 Allen Street, Brockton.
Middlesex University School of Medicine, 1938. Sponsor: Edward B. Gilmore, 232 Main Street, Brockton.

PINZINO, CHARLES J., 722 Main Street, Brockton.
Kansas City University of Physicians and Surgeons, 1940. Sponsor: Alphonse F. Budreski, 704 North Main Street, Brockton.

SHAPIRO, HARRY, 64 Seaver Street, Stoughton.
Middlesex University School of Medicine, 1931. Sponsor: Edward H. Ewing, 15 Capen Street, Stoughton.

SPADEA, SAVERIA, 16 Oakland Place, Brockton.
Loyola University School of Medicine, 1938.

ZALON, LEO, 85 Broad Street, Bridgewater.
Middlesex University School of Medicine, 1931. Sponsor: William E. Hunt, 120 Main Street, Bridgewater.

Ralph C. McLeod, *Secretary*
Goddard Hospital, Brockton

SUFFOLK DISTRICT

BOURGEOIS, GEORGE A., 497 Beacon Street, Boston.
Tufts College Medical School, 1937.

CLOUGH, JOSEPH M., 500 Boylston Street, Boston.
Jefferson Medical College, 1936.

COMPTON, DAVID W., 23 Chambers Street, Boston.
University of Pennsylvania School of Medicine, 1941.

GIANTURCO, NICHOLAS D., 198 Orient Avenue, East Boston.
Middlesex University School of Medicine, 1934. Sponsor: Aubrey C. Benjamin, 35 Eleanor Street, Chelsea.

GOLD, ABRAHAM, 50 Nichols Street, Chelsea.
Middlesex University School of Medicine, 1937. Sponsor: George Reinherz, 270 Chestnut Street, Chelsea.

HAASE, FERDINAND, JR., Massachusetts General Hospital, Boston.
Harvard Medical School, 1942.

HYFER, HARRY J., 38 Summit Avenue, Winthrop.
Middlesex University School of Medicine, 1937. Sponsor: Maurice O. Belson, 311 Commonwealth Avenue, Boston.

KARP, I. ALBERT, 42 Crescent Avenue, Chelsea.
Middlesex University School of Medicine, 1936. Sponsor: Maurice A. Gilbert, 162 Shurtleff Street, Chelsea.

KIRSH, DAVID, 42 Mt. Vernon Street, Boston.
University of Pennsylvania School of Medicine, 1940.

LEVENSON, STANLEY M., Boston City Hospital, Boston.
Harvard Medical School, 1941.

LEVINE, MILTON, 1543 North Shore Road, Revere.
Middlesex University School of Medicine, 1937. Sponsor: Harold L. Musgrave, 620 Beach Street, Revere.

LICATA, DANIEL D., 8 John Avenue, Revere.
Middlesex University School of Medicine, 1937. Sponsor: Harold L. Musgrave, 620 Beach Street, Revere.

MAISLEN, ARTHUR A., 407 Huntington Avenue, Boston.
University of Vermont College of Medicine, 1936.

MILLER, HAROLD, 48 Addison Street, Chelsea.
Midwest Medical College, 1937. Sponsor: Aubrey C. Benjamin, 35 Eleanor Street, Chelsea.

PIPI, JOHN, 821 Saratoga Street, East Boston.
Middlesex University School of Medicine, 1935. Sponsor: Pasquale Costanza, 238 Maverick Street, East Boston.

SHANDS, HARLEY C., 99 Myrtle Street, Boston.
Tulane University of Louisiana School of Medicine, 1939.

WEISMAN, AVERY D., 829 Beacon Street, Boston.
University of Michigan Medical School, 1940.

WEISS, WILLIAM A., Massachusetts General Hospital, Boston.
Jefferson Medical College, 1938.

Robert L. Goodale, *Secretary*
330 Dartmouth Street, Boston

WORCESTER DISTRICT

ABODEELY, ROBERT A., 5 Anna Street, Worcester.
Boston University School of Medicine, 1939.

ACHIN, JOFFRE V., Masonic Home Road, Charlton City.
Cornell University Medical College, 1941.

ALLEN, ROGER E., 653 Main Street, Shrewsbury.
Tufts College Medical School, 1940.

BENNETT, ROBERT E., 463 Salisbury Street, Worcester.
Tufts College Medical School, 1940.

BILLER, SAMUEL B., 252 May Street, Worcester.
Boston University School of Medicine, 1935.

BOLDUC, ROBERT A., 358 Lincoln Street, Worcester.
Tufts College Medical School, 1939.

CARLETON, WILLIAM T., 40 Whitman Road, Worcester.
Harvard Medical School, 1939.

CROSKERY, WILLIAM F., 390 Main Street, Worcester.
Boston University School of Medicine, 1937.

EYRES, ALFRED E., Worcester State Hospital, Worcester.
State University of Iowa College of Medicine, 1932.

FUCHS, JAKOB, 51 School Street, Milford.
University of Vienna, 1923. Sponsor: Joseph Ashkins, 36 Pine Street, Milford.

HAMBERGER, HERBERT, 190 Institute Road, Worcester.
University of Pisa, 1936. Sponsor: Bardwell H. Flower, Worcester State Hospital, Worcester.

HANNON, HARLAND N., 201 Worcester Road, North Grafton.
Kansas City University of Physicians and Surgeons, 1939. Sponsor: Foster L. Vibber, 27 Elm Street, Worcester.

JACOBS, NATHAN, 211 Main Street, Fisherville.
Middlesex University School of Medicine, 1934. Sponsor: Charles N. Church, 117 Elm Street, Millbury.

KAPLAN, DANIEL, 1006 Pleasant Street, Worcester.
Middlesex University School of Medicine, 1936. Sponsor: Adolph Meltzer, 16 Norwich Street, Worcester.

KARP, LEWIS S., 460 Park Avenue, Worcester.
Middlesex University School of Medicine, 1937. Sponsor: Joseph W. O'Connor, 36 Pleasant Street, Worcester.

KRETZMER, EUGENE, 54 South Lenox Street, Worcester.
University of Munich, 1908. Sponsor: Ernest L. Hunt, 28 Pleasant Street, Worcester.

MULCAHY, RICHARD E., 22 Capron Street, Uxbridge.
Tufts College Medical School, 1940.

OLIVER, HOWARD M., 25 Catharine Street, Worcester.
Jefferson Medical College, 1944.

REARDON, EDWARD R., 32 Melrose Street, Worcester.
Tufts College Medical School, 1937.

ROTHSCHILD, ALFRED F., 87 Richmond Avenue, Worcester.
University of Munich, 1918. Sponsor: Joseph W. O'Connor, 36 Pleasant Street, Worcester.

SCANLON, JAMES G., Memorial Hospital, Worcester.
Hahnemann Medical College and Hospital of Philadelphia, 1942.

WARREN, JOSEPH E., 425 Salisbury Street, Worcester.
Harvard Medical School, 1938.

Leslie P. Leland, *Secretary*
57 Cedar Street, Worcester

WORCESTER NORTH DISTRICT

DIAMOND, MORRIS, 2 Myrtle Street, Athol.
University of Vienna, 1920. Sponsor: Donald B. Cheetham, 164 Exchange Street, Athol.

GASPAR, SHARI B., Hospital Cottages for Children, Baldwinsville.
Royal Hungarian University of Pecs, 1925. Sponsor: Edwin St. J. Ward, Templeton.

PARNES, JACOB, Waltham Regional Hospital, Waltham (formerly: Leominster).
University of Prague Medical Faculty, 1937. Sponsor: Bartholomew P. Sweeney, 5 Gardner Place, Leominster.

SCHECHTMAN, HAROLD I., Fort Devens, Ayer.
Middlesex University School of Medicine, 1934. Sponsor: Joseph D. Quinlan, 44 Prichard Street, Fitchburg.

James G. Simmons, *Secretary*
30 Myrtle Avenue, Fitchburg

MEDICOLEGAL ABSTRACT

Regulation of Practice by Government: Qualifications to Practice. A man who had taken out life insurance from the defendant company died. The plaintiff was his beneficiary. The policy contained a clause which provided that, if the insured had received medical or surgical treatment within two years prior to his application for insurance and had not disclosed this fact on the application, the policy would be voidable. The deceased did receive treatments from a chiropractor within the period and did not disclose this fact.

The beneficiary brought suit to recover the proceeds of the policy, which the company refused to pay on the grounds that the treatment by the chiropractor was undisclosed medical or surgical treatment rendering the policy voidable. The trial court refused to direct a verdict for the defendant, and the defendant appealed. In reversing the judgment, the Supreme Court of New Jersey said that the Legislature had regulated the practice of chiropractic under "an act to regulate the practice of medicine and surgery" and that it was within the power of the Legislature to so classify it.—(*Kahn* v. *Metropolitan Life Insurance Company*, 41 Atl. [2d] 329, March 1, 1945, New Jersey.)

In New York, however, possession of a mere license to practice podiatry or chiropody, without more, does not confer on the licensee the right to call himself "doctor."

Originally the educational requirements to practice podiatry and chiropody were one year of college and three years of professional training. Subsequently they were increased, and graduates were admitted to the degree of doctor of podiatry or chiropody. This placed those who were licensed under the original requirements at a disadvantage, and they sought to call themselves "doctor" too. They therefore brought an action seeking to have rescinded a rule promulgated by the Regents of the State University declaring the use of the title "doctor" by those who had not fulfilled the more recent requirements a fraud and a deceit that would subject the offender to disciplinary action.

The court found that the delegation by the Legislature to the Regents of the authority to make such rules was proper and that, in the absence of action on the subject by the Legislature, the Regents could make the rule in question. — (*Buhl* v. *University of State of New York*, 52 N.Y.S. [2d] 511, Dec. 29, 1944.)

CORRESPONDENCE

CARDIAC MURMURS

To the Editor: In view of the enormous amount of unnecessary invalidism and psychic trauma caused by physicians who are unable to evaluate systolic murmurs, it seems unfortunate that Wolfson's communication, "Clinical and Laboratory Studies of Ninety-One Workers with Apical Systolic Murmurs," which appeared in the December 20, 1945, issue of the *Journal*, did not elicit at least a modifying editorial comment.

The author states, "A murmur was considered to be associated with a cardiovascular abnormality when the patient had a history of rheumatic fever [a doubtfully valid assumption], hypertension, roentgenologic evidence of cardiac enlargement, an abnormal electrocardiogram, or other disease that might be expected to produce a murmur [again, a doubtful assumption unless one considers the hemic murmur of anemia to be evidence of a cardiovascular abnormality]."

The author also states: "In the youngest age group, below twenty-five, a history of rheumatic fever was obtainable in more than half the cases. . . . From this it may be concluded that an apical systolic murmur in a young patient is likely to be associated with rheumatic fever. . . ." On going back to the author's criteria for a past history of rheumatic fever, however, one finds the following statement: "Careful inquiries were made for a past history of rheumatic infection, and this was said to be positive only when it included at least one of the following: an illness characterized by swollen, painful, tender joints; an illness of several weeks' duration, diagnosed at the time by the attending physician as rheumatic fever; *prolonged bed rest recommended by a physician because of the discovery of a heart murmur* [italics mine]; and St. Vitus's dance." It seems most unfortunate that the author accepts "prolonged bed rest recommended by a physician because of the discovery of a heart murmur" as evidence of rheumatic infection. I believe that this assumption is entirely unjustified and, as often as not, represents an incorrect diagnosis on the part of the attending physician.

Judging from personal experience in a small outpatient cardiac clinic (Cambridge Hospital), on the medical ward service of a large city hospital (Boston City Hospital) and in the internists' department at an induction center (First Service Command), I believe that the finding of an apical systolic murmur as the sole sign of a cardiac disorder in a person under twenty-five without hypertension, hyperthythyroidism, tachycardia, fever, anemia or a clear-cut history of rheumatic fever is better ignored than studied.

This, I believe, is still true even in the presence of organic heart disease. An apical systolic murmur in the absence of cardiac enlargement (by x-ray) or diminished exercise tolerance is no contraindication to normal physical activity, including strenuous sports, and should never be the reason for putting a person to bed for weeks or months.

The incidence of cardiac neuroses and unnecessary cardiac invalidism in young people with systolic murmurs is a most unfavorable commentary on the practicing physicians in this country. The stethoscope is a double-edged sword, and I still agree with MacKenzie that the physician would do well to throw away his stethoscope while evaluating cardiac murmurs.

DUDLEY MERRILL, M.D.

50 Brattle Street
Cambridge, Massachusetts

NOTICES

ANNOUNCEMENTS

Dr. William W. Babson announces his return to the practice of surgery at 79 Prospect Street, Gloucester.

Dr. James R. Corkery, having returned from military service, has resumed practice at 6 Hancock Street, Everett.

Dr. Sidney Derow, having been released from active duty with the United States Navy, has resumed his practice at 925 Centre Street, Newton Centre.

Dr. Clifford C. Franseen announces his return from military service and the reopening of his offices at 1180 Beacon Street, Brookline, for the practice of surgery.

Dr. Irvin G. Gahm announces his return from military service and resumption of practice at 414 Walnut Street, Newtonville.

Dr. Stuart N. Gardner, having returned from service with the United States Navy, announces the reopening of his office at 24 Chestnut Street, Salem, for the practice of internal medicine.

Dr. A. Gordon Gauld announces the reopening of his office for the practice of obstetrics and gynecology at 330 Dartmouth Street, Boston.

Dr. Richard J. Gorman, having returned from service with the United States Navy, announces the opening of his office at 628 Centre Street, Jamaica Plain, for the general practice of medicine.

Dr. Lyman H. Hoyt, having returned from service in the Medical Corps of the United States Navy, has resumed his practice at 51 Bay State Road, Boston.

Dr. Samuel W. Joel announces his return from military service and the reopening of his office for the practice of psychiatry and neurology at 510 Commonwealth Avenue, Boston.

Dr. William E. Ladd announces the removal of his office from the Children's Hospital to 330 Dartmouth Street, Boston.

Dr. William N. Lanigan, recently released from military service, announces the reopening of his office at 187 Main Street, Medford, for the practice of orthopedic surgery.

Dr. Charles C. Lund announces the removal of his office from 319 Longwood Avenue to 20 Gloucester Street, Boston.

Dr. Charles J. McCarthy has been released from military service and has resumed the practice of medicine at 466 Medford Street, Somerville.

Dr. John D. Shinberg, having returned from the armed services, announces the reopening of his office at 54 Merrimack Street, Haverhill.

MEDICAL LIBRARY ASSOCIATION

The Medical Library Association will hold its postwar convention at the Yale Medical Library, New Haven, Connecticut, on March 25, 26 and 27. The program will include papers on the history of medicine in Connecticut by Drs. Herbert Thoms and Harold S. Burr and on the libraries of Yale University by Mr. J. T. Babb, Dr. John F. Fulton and others. Dr. Sanford V. Larkey will speak on his experiences as medical historian of the European Theater. Miss Ethel Wigmore, of the National Institute for Medical Research, will discuss British and Continental medical libraries. There will also be reports on the organization of the Army Medical Library by members of its staff and general discussions on interlibrary and international co-operation. The training necessary for medical librarianship is the subject chosen for the presidential address by Miss Mary Louise Marshall. The speaker at the annual banquet will be Professor C.-E. A. Winslow. Headquarters will be at the Hotel Taft. Further details may be obtained from the secretary — Miss Frida Pliefke, 2000 Medical Arts Building, Minneapolis, Minnesota.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Thursday, March 14

Friday, March 15

*9:00–10.00 a.m. Clinicopathological Conference. Drs. Chester S. Keefer and H. E. MacMahon. Joseph H. Pratt Diagnostic Hospital.

*10:00 a.m.–12:00 m. Medical Staff Rounds. Peter Bent Brigham Hospital.

10:50 a.m. Ultraviolet Radiation Therapy in Diseases of the Skin. Dr. Edward LaFreniere. (Postgraduate Clinic in Dermatology and Syphilology.) Amphitheater, Dowling Building, Boston City Hospital.

Monday, March 18

*12:00 m.–1:00 p.m. Clinicopathological Conference. Peter Bent Brigham Hospital.

Tuesday, March 19

*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

Wednesday, March 20

*9:00–10:00 a.m. Diagnostic Errors in the Pratt Hospital. Dr. C. B. Popplestone. Joseph H. Pratt Diagnostic Hospital.

*10:30–11:30 a.m. Medical Clinic. Isolation Building Amphitheater. Children's Hospital.

*12:00 m. Clinicopathological Conference (Children's Hospital), Amphitheater, Peter Bent Brigham Hospital.

*2:30–4:00 p.m. Combined Clinic by the Medical, Surgical and Orthopedic Services. Amphitheater, Children's Hospital.

*Open to the medical profession.

January 7–April 22, 1946. Metropolitan State Hospital. Eleventh postgraduate seminar in neurology and psychiatry. Page 314, issue of September 6.

February 4–March 29. Health Education Institute. Page 746, issue of December 13.

March 11. Massachusetts Hospital Association. Page 240, issue of February 14.

March 12. Greater Boston Medical Society. Page 312, issue of February 28.

March 12. Harvard Medical Society. Amphitheater, Peter Bent Brigham Hospital. 8:00 p.m.

March 14. Bursitis at the Shoulder Joint: Diagnosis and treatment. Drs. Francis C. Hall and Robert B. Osgood. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

March 15–September 15. Boston University Course for Discharged Medical Officers. Page 240, issue of February 14.

March 25–27. Medical Library Association. Notice above.

April 1–June 1. Intensive Course in Ophthalmology. Page 240, issue of February 14.

April 4. Hermann M. Biggs Memorial Lecture. Page 206, issue of February 7.

May 13–17. American College of Physicians. Page 798, issue of December 20.

June 20–22. American Association for the Study of Goiter. Page 312, issue of February 28.

District Medical Society

WORCESTER

March 13. Worcester Memorial Hospital.
April 10. Hahnemann Hospital.
May 8. Annual meeting.